

BRISTOL  
ROYAL INFIRMARY  
REPORTS.

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Vol. I.—1878-79.

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ROYAL INFIRMARY  
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*Edited by*

W. H. SPENCER, M.A., M.D.,

AND

J. GREIG SMITH, M.A., C.M.

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## ERRATA.

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P. 72, line 9 from top, *for* "spaec" *read* "space."

P. 95, line 2 from top, *for* "absorbitive" *read* "absorptive."

P. 240, line 1 from top, *for* "construction" *read* "constriction."

Pl. XI. (P. 98) Fig. 2. Granular condition of bone over-represented, and carried too high up; club-shaped masses too long, and not deeply enough shaded.



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## ACUTE MYELITIS, AS ILLUSTRATING THE PHYSIOLOGY OF THE SPINAL CORD.

BY DR. E. LONG FOX.

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THE study of myelitis will show that it serves as a connecting link between several lesions of the spinal cord that seem at first sight scarcely to belong to it. The most usual starting point is the grey matter, and over a large extent the lesion may be confined exclusively or almost exclusively to it, "central softening": or the changes may be limited to the immediate neighbourhood of the central canal: or the whole thickness of the cord may be pretty generally affected, in which case the longitudinal extent is less "acute myelitis strictly": or the lesion may be of greater longitudinal extent, but affecting only the circumference of the cord, in which case it is generally associated with primary or secondary inflammation of the membranes, "acute perimyelitis and meningo-myelitis." There is also met with a circumscribed softening, small, disconnected, at various heights and in various parts of the cord, both in the white and grey matter, "Disseminated Softening." Myelitis is the starting point of Infantile Paralysis, and of Spinal Paralysis of adults. In its more chronic developments it may lead to sclerosis, and thus may be considered as the original cause of those phenomena which depend either on Insular Sclerosis, on sclerosis of the Posterior Columns, or of the antero-lateral columns: and the atrophy of the cells of the anterior horns, associated with the

symptoms of Progressive Muscular Atrophy, owns as its first stage an inflammation of this portion of the grey matter.

Of no less importance is the variation in the degree of this morbid condition. So many degrees come before us in private practice, and life is often so protracted in these cases, that their true pathology can only be recognised in the wards and deadhouse of an Hospital.

According to the anatomical change the disease is separated into two groups, acute and chronic. It attacks the grey matter most frequently. It is generally circumscribed; more rarely it is diffused over the whole length of the cord. It may terminate either in one of the various forms of inflammatory softening, the more usual termination, or very exceptionally in abscess, or in destruction of the nerve elements without softening, "Acute hyperplastic Myelitis."

These modes of termination suggest therefore a natural division of acute myelitis into the form accompanied by softening, "Acute Myelo-malacia," and the form without softening "Acute hyperplastic Myelitis." This division is the more desirable, in that somewhat different regions in the thickness of the cord are affected in these two forms, and thus some variation in symptoms is the necessary consequence.

The lesions occur either spontaneously, or from compression: in the latter case it is most frequently associated with injury or disease of some one or more vertebræ, fracture, dislocation of vertebræ (very rare), dislocation of a portion of a ruptured intervertebral disc, caries, or necrosis (rare): but it may be the sequence of pressure within the bony canal, from tumour of membranes, or of the cord itself, or from the various forms of spinal apoplexy, extra-



membranous, intra-membranous, or within the substance of the cord itself. In one case in the Infirmary myelitis ran a subacute course for several months, and the lesions found were necrosis of the body of the tenth dorsal vertebra, the cord creamy from the eighth to the eleventh dorsal vertebra, and the cauda equina in a state of sphacelus. At the seat of softening all the antero-lateral columns were composed of indurated and proliferated connective tissue. The central grey matter and the posterior columns were softened.

Taking first the variety accompanied with softening, there are three forms usually met with, that only imperfectly represent the various stages of the disease, red, yellow, and grey softening, more reasonably named by some French authors the stages of swelling, of true softening, and of disintegration or absorption.

In the stage of red softening, the parts affected are swollen by hyperœmia and œdema. There are often minute extravasations. The nerve fibres are separated by the extravasated blood. Microscopically these extravasations are seen to be undergoing changes. There are frequently small circumscribed spots of granular exudation about the vessels. Round the vessels and in the interstitial tissue an infiltration of blood, more or less changed, may compress the nerve elements, and even in this stage lead to their complete disintegration. In the neuroglia we find division of nuclei and proliferation of the cell elements of the tissue; and, as this stage is passing into the second, the formation of granular cells, originating partly from divisions of the neuroglia cells, partly from the destruction of nerve fibres (Pl. II., fig. 1). The preserved nerve fibres seem sometimes thinned and atrophied (Pl. II., fig. 2), sometimes enlarged by imbibition from soaking

### EXPLANATION OF PLATE I.

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Fig. 1.—*Ascending degeneration affecting the posterior columns.*

Fig. 2.—*Apparent increase of the size of the medullary sheath of nerve tubes. Proliferation of Neuroglia.*

Fig. 3.—*Swollen nerve fibres.*

Fig 1.  $\times 40$



Fig 2  $\times 300$

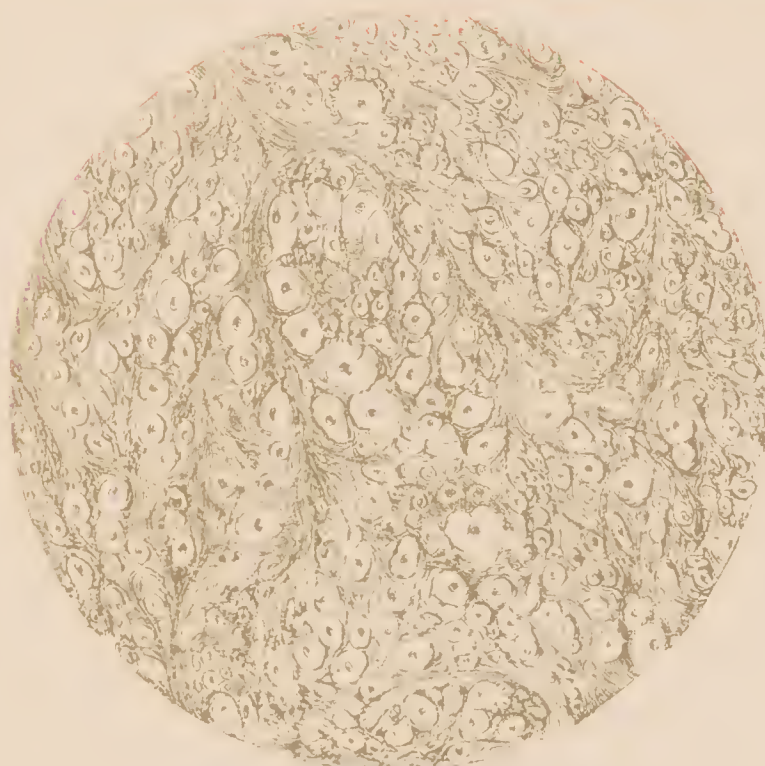
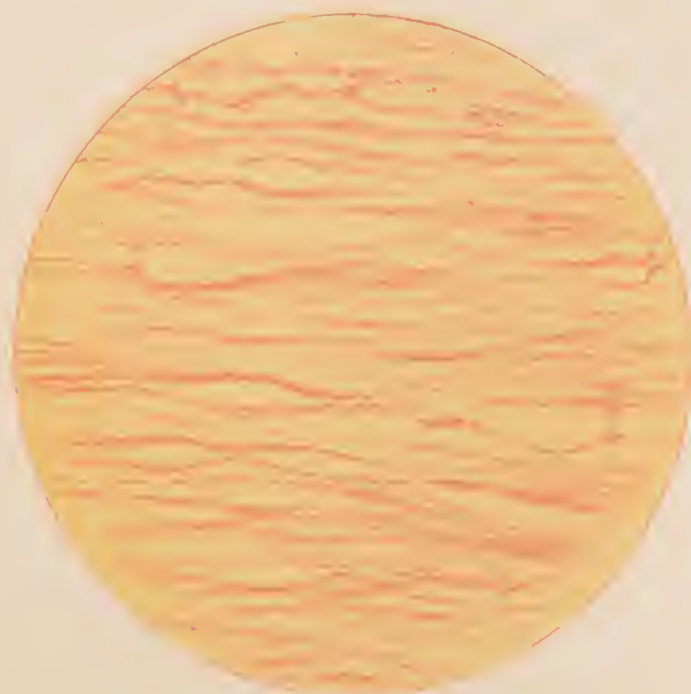


Fig 3  $\times 300$







(Pl. I., fig. 3), this enlargement often only occurs here and there in a nerve fibre (Pl. II., fig. 1). The axis-cylinders are swollen, and the myeline itself atrophied almost to the point of extinction. Some observers have found similar changes in the bodies of the nerves, enlargement and swelling of cells, swelling of nuclei, division of nuclei, and later on the commencement of fatty degeneration, or true atrophy.

In the second stage fatty degeneration predominates. The yellow colour of the softened parts depends partly on the change of the colouring matter of the blood, partly on fatty degeneration. The development of granular cells increases, drops of fat appear, an advancing degeneration obtains of the nerve elements and of the connective tissue. According to the amount of bloody or purulent infiltration the colour varies from white and yellow to reddish-brown or red. The consistence may vary from slighter degrees of softening to pulpy deliquescence. Microscopically there is an accumulation of granular cells and of fat drops. Granular pigment appears, especially in the ganglionic cells (Pl. IV., fig. 2). In this stage, too, there is here and there some thickening of vessels, and hypertrophy of the connective tissue (Pl. II., fig. 3). This tissue also shows commencing softening and degeneration. There is advancing degeneration of the nerve fibres and the ganglionic cells. In other parts there is still further progress toward death of the part, spots of softening of greater or smaller extent, pus globules and debris of more or less changed nerve substance.

The third stage may never be reached. It may be, that before things get to that pass, some process of repair is set up. When this is not the case, the parts previously swollen become attenuated, and the colour changes to a

EXPLANATION OF PLATE II.

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Fig. 1.—*Swollen nerve fibres. Large conglomerate masses. Incipient myelitis in overdriven ox.*

Fig. 2.—*Enlarged vessels. Swollen and atrophied tubes. Amyloid bodies.*

Fig. 3.—*Thickening of vessels. Great increase of connective tissue growth.*

Fig 1.  $\times 300$

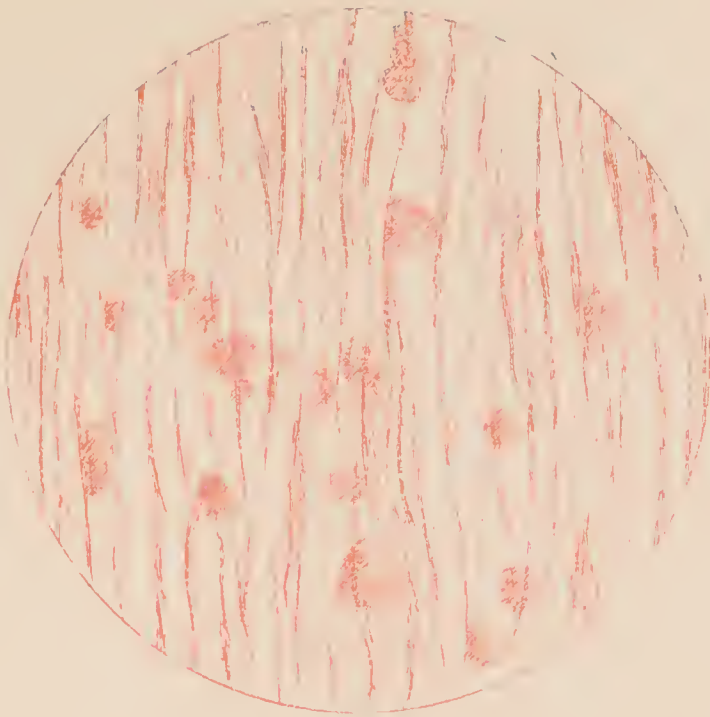


Fig 2.  $\times 300$

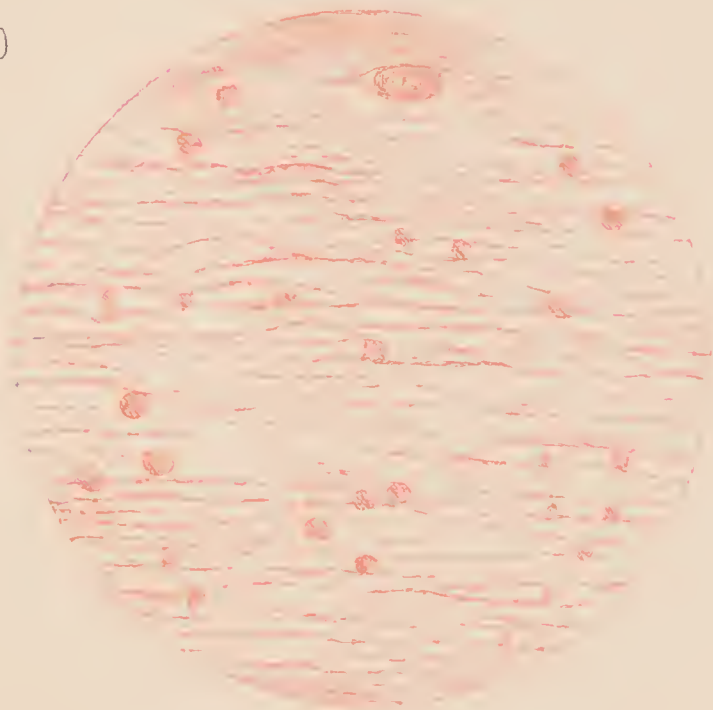
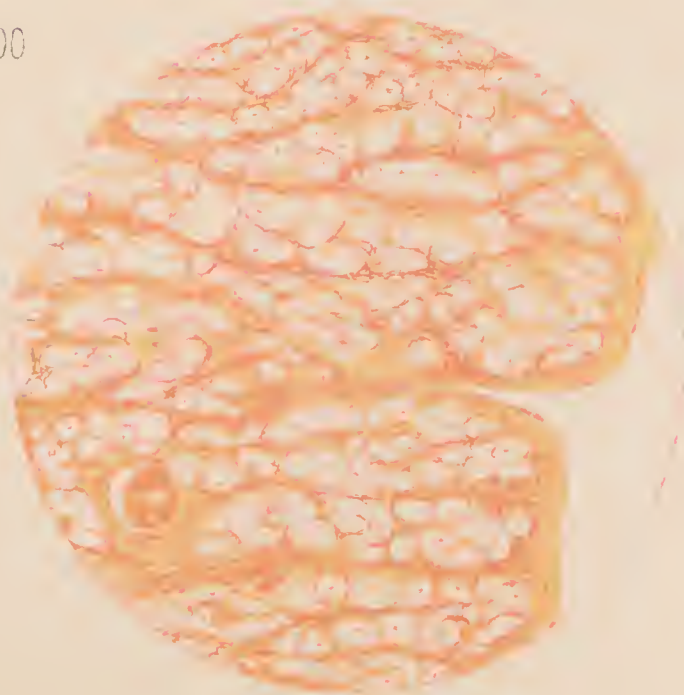


Fig 3.  $\times 300$







white or a whitish grey. The diminution in size is caused partly by a gradual absorption of the infiltration or of the liquefied tissue, partly by a decrease in the granular cells, and by the advanced decay of nerve elements, and chiefly perhaps by the contraction of the hypertrophied connective tissue. The consistence, too, of the diseased part at this stage depends very much on the condition of this tissue. If the connective tissue has not been much implicated (a rare event) the consistence may be creamy, from the softening and only partial absorption of the nerve elements. On the other hand the liquefied debris of nerve elements may be often absorbed almost entirely, and there will remain a zone of thickened membranes, and within them spots of indurated tissue produced by the contraction of the morbid connective tissue, and reaching more or less completely the condition of sclerosis (Pl. III., figs. 1, 2 and 3; Pl. IV., fig. 3). Another termination is a mingling of these two; a formation of small cavities filled with fluid, and pierced through with thickened connective tissue. It is evident however that this is only the apparent condition; the rationale being inflammation of nerve elements, and of connective tissue, liquefaction and partial absorption of the former, and contraction of the latter to form walls and thickened trabeculæ to these cavities.

When abscess occurs (very rare) it takes some such form as this, occurring in limited and encapsulated spots, the capsules consisting of this same contracted connective tissue.

In a very general sense the symptoms correspond to the variety of the lesion. Severe symptoms may be developed very suddenly in the most acute form of acute myelitis. In bad cases the most marked phenomena appear within the first few days, and in the suddenness of

EXPLANATION OF PLATE III.

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Fig. 1.—*Atrophy of ganglion cells.*

Fig. 2.—*Myelitic softening : stage just preceding absorption.*

Fig. 3.—*Atrophy from myelitis. Thickening of pia mater.*

Fig 1 x 300.

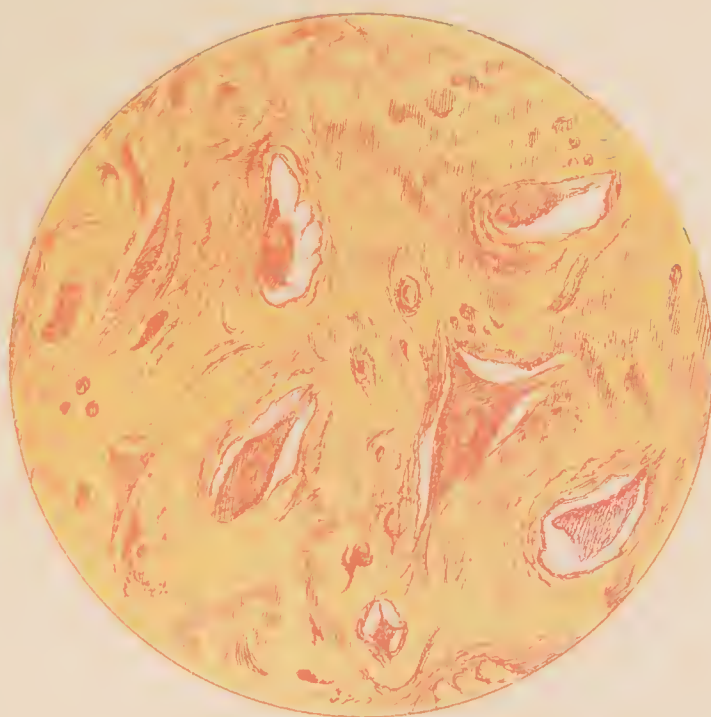


Fig 2 x 300

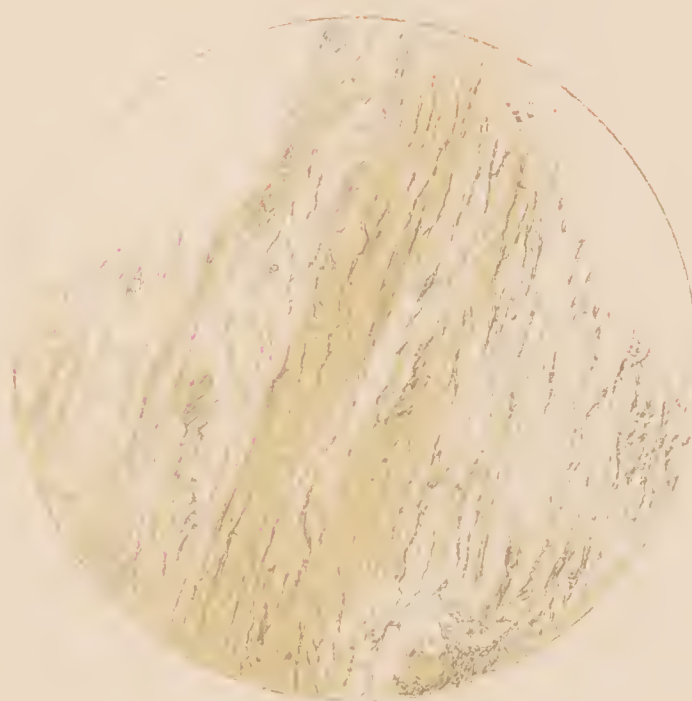
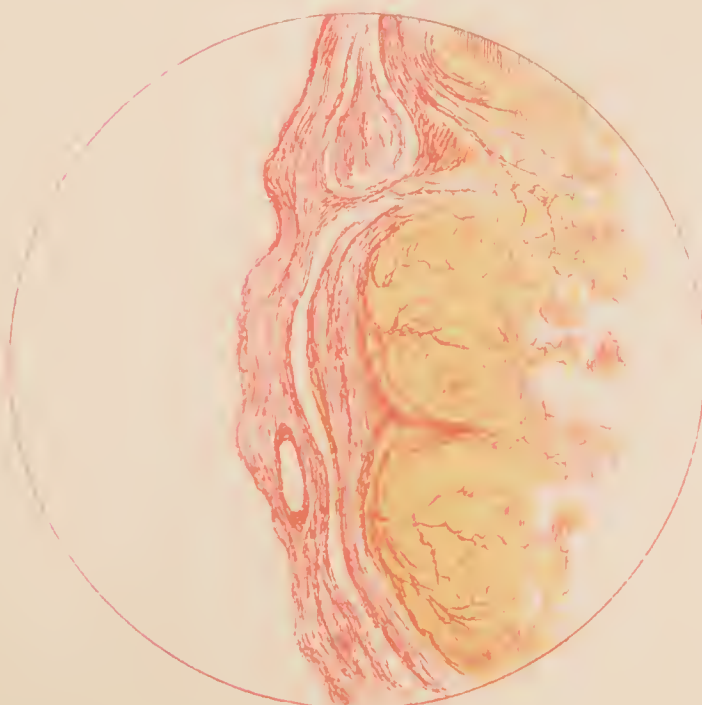


Fig 3. x 300.





their onset will resemble spinal apoplexy. In other cases days and weeks may pass before paralysis is complete; and this symptom will be preceded by sensory or motor phenomena of irritation, lancinating pains, paraplegiæ, muscular spasm, cramp or convulsion, set up spontaneously or by a reflex act, especially in the lower extremities, and the paralysis manifests itself gradually in the lower parts of the body, affects one part after another in succession and with unequal intensity, advancing from one muscle-group to another, from one extremity to its fellow.

The great variation in the position of the softened patches in the cord, both transversely and longitudinally, leads to a proportionate variation in the distribution of the motor paralysis, the loss of power taking the form of spinal hemiplegia, or hemi-paraplegia, or, again, of diplegia or monoplegia of the upper extremities, if the lesion is situated in the cervical region.

The disturbances of sensation are less prominent, except when the softening is a very rapid consequence of violent injuries to the bony structure. In these cases sensory paralysis is often synchronous with motor (the lesion being pretty uniformly bilateral), but seldom extends quite so high up in the body as the latter; and if life is prolonged for a time the loss of sensation is usually a little behind the loss of motion. In very localised lesions, however, with spinal hemiplegia, there may be hyperæsthesia of the paralysed side, with anæsthesia of the non-affected side, as in animals whose cord is divided longitudinally in the middle line.

The second form, acute hyperplastic myelitis, poliomyelitis anterior acuta, serves as a connecting link between the more acute and the more chronic forms of inflammatory disease of the cord.



EXPLANATION OF PLATE IV.

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- Fig. 1.—*Thickening of neuroglia. Atrophy of nerve substance.*
- Fig. 2.—*Pigmented and degenerated ganglion cells of anterior horns.*
- Fig. 3.—*Coats of vessels thickened. Great degeneration of grey matter.*



Fig. 1  $\times 300$



Fig. 2  $\times 300$



Fig. 3  $\times 300$





This variety of myelitis is not associated with softening except in its early stages. It affects mainly, with scarcely any extension into the neighbouring parts, the anterior grey substance, and especially of the cervical and lumbar bulbs. The anterior cornua are at first hyperæmic, become soft, infiltrated with granulation cells and numerous nuclei, and the connective tissue is increased and thickened. Very rapidly many of the large ganglionic cells of the anterior cornua degenerate and become atrophied, and even disappear altogether, or the atrophied cells lie side by side with others that are normal. Later on the granulation cells are no longer seen, but the seat of the lesion is occupied with numerous corpora amylacea. The anterior roots are frequently atrophied.

It is probable that the disease is from the first interstitial, the connective tissue being primarily affected; and this newly-formed tissue may take on fatty degeneration, or may lead to the formation of cysts, or, as it contracts, may approximate more or less closely to the true sclerosis met with in chronic myelitis (Pl. IV., fig. 1; Pl. I., fig. 2).

Clinically this variety of myelitis lies at the foundation of those diseases which we distinguish as infantile spinal paralysis and acute or sub-acute spinal paralysis of adults.

Eulenburg thinks that there should be included under this form isolated cases of functional paralysis, and of paralysis and ataxy occurring after acute diseases, though most of such cases should more properly be referred to a disseminated chronic myelitis, "*Sclérose en plaques.*"

As a sequence of this lesion the muscles degenerate partly into fatty, partly into connective tissue; the bones are retarded in their growth both in length and thickness, the joints become lax from erosion of the articular extremities, atrophy of cartilages and degeneration of ligaments,

and as a result of all this the patient becomes deformed in many ways.

The special phenomena attending these lesions are fever of very temporary duration, various cerebral symptoms, of which in children convulsion is the most prominent, rapid paralysis, followed by atrophy of the paralysed muscles, either of one or more groups of muscles, or of a limb or several limbs at once, with incomplete repair, with early absence of reflex irritability, electrical evidences of degenerative change, namely, early extinction of faradic excitability of the affected muscles and of galvanic irritability of the nerves, whilst at first there is some increase of galvanic irritability in the muscles, with peculiar slowness of contraction, and eventually a great diminution. Sensation is perfect throughout, if the lesion affects only the anterior cornua, and the bladder disturbances are too slight and transient to be worth notice. The late deformities are due to the paralysis, the action of antagonist muscles, the muscular atrophy, the arrest of bone development, the trophic disturbances of the joints and the weight of the body itself; all these causes combining to produce the various deformities of the foot, the knee and the spinal column, which are so remarkable in the spinal paralysis of children. Amongst the more interesting phenomena of this disease may be mentioned the coldness and change of colour in paralysed limbs from vaso-motor paralysis, and the fact that not unfrequently certain muscles either stand aloof from the morbid action or alone in the limb are subjects of it. Amongst these the Deltoid, the Tibialis anticus, the Sartorius, the Supinator longus may be cited as examples.

In the spinal paralysis of adults the sexual function is normal, and the deformities are less.



A word must be said about Chronic Myelitis. The connection between this form and the preceding is tolerably close, from the fact that probably all forms of chronic myelitis are in their early stage of an interstitial nature. The so-called parenchymatous myelitis and the true interstitial myelitis can only be looked upon as different consequences of one common cause—chronic interstitial inflammation,—although Charcot considers that in parenchymatous myelitis the nerve-cells are attacked first and the neuroglia only later.

The question as to whether every chronic myelitis is a sclerosis, and every sclerosis a chronic myelitis, may be answered thus:—Every sclerosis is the result of chronic inflammatory action, but it is the last result. It means not only proliferation of connective tissue fibres, but retraction of them. Chronic inflammation may be hindered in its course by remedies. It may originally be extremely sub-acute; it may proceed up to a certain point and no farther from a variety of unknown causes, and may never attain to sclerosis, so that the two terms are not absolutely convertible.

The minute pathological anatomy of chronic myelitis has little to do with the subject of this paper, but some of its forms are so closely connected with the more acute varieties of myelitis that they must be mentioned. Chronic myelitis, like acute myelo-malacia, shows regional variations, diffuse or circumscribed, longitudinal or transverse, continuous or disseminated.

1. The form called multiple, circumscribed or disseminated sclerosis is generally associated with similar lesions in the brain, and its course and phenomena mainly depend on this connection.

2. Of the forms confined to the cord we note next the



chronic inflammations of the cord, called by Charcot and Hallopeau the parenchymatous forms of chronic myelitis.

Of these (I.), sclerosis of the posterior columns, the usual lesions of *Tabes Dorsalis*, *Progressive Locomotor Ataxy*.

II. Sclerosis of the lateral columns, *Spasmodic Tabes Dorsalis*, *Spastic Spinal Paralysis*.

III. Lateral amyotrophic sclerosis, sclerosis of the lateral columns with implication of the anterior horns.

IV. Chronic inflammation of the anterior horns, *Polio-myelitis anterior chronica*, which, in the opinion of some authors, embraces the lesion in lead poisoning. Eulenberg places under this head *Progressive Muscular Atrophy* and *Pseudo-hypertrophy of Muscles*; but the fact that in *Poliomyelitis anterior chronica* the atrophy follows the paralysis, whereas in *Progressive Muscular Atrophy* it precedes it, renders this arrangement doubtful.

3. There remain those forms of chronic interstitial myelitis of the French authors, or of sclerosis in a narrow sense, which are restricted to the cord, and occur there either in large circumscribed spots or as a diffused condition, "*Sclérose en plaques*" and "*Sclérose diffuse*."

We must specially distinguish those forms of sclerosis which only occur at the circumference of the cord and are associated with disease of the membranes, "*Chronic Perimyelitis*" or "*Myelo-Meningitis*"; and those confined to the neighbourhood of the central canal "*Chronic central Myelitis*," "*Sclérose periependymaire*," in which the central canal is often narrowed or obliterated either by a connective tissue with fine granules, and containing vessels, or by collections of cells rich in nuclei and also containing vessels. To this division, too, belong those cases of formation of cavities, and perhaps of so-called

doubling of the central canal, caused probably by the liquefaction of the masses which filled the canal and the formation of a single or double lumen in it. Avoiding all reference to the symptoms of chronic myelitis, there remain traumatic softening of the cord, compression myelitis, spontaneous acute or sub-acute softening of the cord, acute hyperplastic myelitis, over circumscribed or disseminated regions, abscess of cord and acute myelomeningitis.

Taking briefly the main symptomatology of some of these varieties, the individual phenomena may be spoken of later. Traumatic softening may affect any portion of the cord, but is more frequently met with in the lower dorsal and lumbar regions than elsewhere. The symptoms vary according to the intensity of the disease. The region of the Sciatic nerve is usually more implicated than that of the Crural. There is therefore complete, or nearly complete, paralysis of the muscles of the posterior portion of the upper thigh and of the lower part of the thigh and foot, whilst there is more or less persistence of motor power in the ileo-psoas and the extensors of the upper thigh. The more complete the paralysis is from the first, the more severe the course of the disease. The sphincters are generally paralysed. Reflex irritability is sometimes raised at first at a very early stage after the injury; but as the softening affects the cord transversely, it rapidly wanes and is wholly lost.

The position of the Anæsthesia corresponds to that of the motor paralysis. It is complete in the posterior portion of the thigh, on the nates, in the neighbourhood of the anus and in the urethra. It is less intense on the upper surface of the thigh. It seems probable that the Anæstheria depends on lesions of different portions of the

diameter of the cord, since by experiment injuries to the posterior columns in the upper lumbar region lead to insensibility to touch on the region of the anus, whilst sensation and motor power may persist in the lower extremities.

If motor power, sensation, and reflex irritability are all impaired, the muscles of the lower extremities become flaccid, weak, and atrophied from implication of the anterior horns; and electric irritability is diminished, and finally destroyed.

In severe cases other trophic and vasomotor disturbances are early met with, decubitus, cystitis, bullæ over the seat of the paralysis, œdema of the feet and legs.

Such cases are very fatal: but life is sometimes prolonged even for years, when, after injury in this region and consecutive myelitis, the paraplegia, the anæsthesia, and the affection of the sphincters persist.

The prognosis depends on the seat and intensity of the lesion, on the existence of positive fracture of vertebræ, and on the course of the disease. Early development of decubitus, cystitis, œdema is grave; as also is the early loss of electric irritability: and more general symptoms, such as collapse, mental depression, loss of appetite and of sleep, all have the same unsatisfactory import.

Compression myelitis is usually slow in its course, with localised pain, or faint paresis of a muscle or group of muscles as a preliminary symptom. The motor and sensory phenomena may advance very slowly for a considerable period, and then a sudden exacerbation of all symptoms take place, rapid softening of cord, and death.

It is the gradual outset that distinguishes this form from the preceding, in which also many of the symptoms are due to myelitis from compression, especially in cases



of fracture of vertebræ, or whenever intraspinal hæmorrhage necessarily leads to compression. But in traumatic cases there are usually the results of spinal concussion, which are wanting in myelitis from slow compression, and the hæmorrhage is frequently enough to induce symptoms of greater severity and extent than appear even after a long time from caries and intra-spinal tumour. Hæmorrhage outside the cord itself may be so limited, that the results of its pressure only affect a single limb or certain groups of muscles, and if meningitis and consequent contraction of the cord is not set up, the hæmorrhage may be absorbed and the paralysed region recover itself.

In spontaneous acute spinal softening (omitting in this place all reference to acute bulbar paralysis, in which the cord is affected over a great portion of its length) two varieties are met with, one in which the inflammation affects a circumscribed portion of the cord, the cervical or more usually the dorsal region, the other in which the disease is more generally diffused throughout the whole cord. The latter form is more frequently developed as a rapid extension of a myelitis originally circumscribed.

The symptoms in this, as in all forms of the disease, vary according to the anatomical extension of the lesion. In the cervical region, if the lesion be too low to affect the phrenic nerve, the paralysis will involve the upper and lower extremity at the same time, but not necessarily on both sides simultaneously. As the upper dorsal is affected, respiration is somewhat difficult, and the feeling of weight in the paralysed limbs and of general weariness is marked. In the lower dorsal region the paraplegia of the lower extremities is often as decided as in severe traumatic forms, but the extension of the disease transversely and its intensity vary so much that the severity of the symp-

toms correspond. The sphincters are usually implicated, and trophic disturbance, loss of reflex and of electric irritability occur; but all the morbid phenomena may be uni-lateral, or at least unequally bi-lateral.

In the diffuse form we find first the symptoms belonging to the region first affected, then a rapid extension upwards, not by implication of Goll's column only, as is the case in ascending degeneration (Pl. I, fig. 1), but of the central grey matter, leading more or less rapidly to trophic disturbances, to implication of the upper extremities, dyspnœa, dysphagia, and, if life is preserved long enough, to some difficulty in articulation.

The symptoms of myelitis are of interest, as illustrating the physiology of the cord.

The motor phenomena predominate in importance, the varied degrees of paralysis, of spasm, and of contraction, the loss and intensification of reflex irritability, the reaction or want of reaction to faradic and galvanic stimulus, and the affection of the sphincters.

The worst cases are those in which the paraplegia is complete, and is accompanied by loss of power over the sphincters and anæsthesia. Such are the cases of which we get *post mortem* records; but it is far more usual to meet with myelitis in a less acute form. The paralysis of the extremities depends on the level in the cord at which the lesion exists. If the arms are early affected, the lesion is at the cervical bulb. Very frequently here only one arm is paretic in the early stages; or the two arms very unequally. The diagnosis however of the position of the lesion should be made from the early symptoms, as many of the less severe cases of myelitis, in which at first the lower extremities are alone implicated, manifest some



paralysis of the upper limbs at a later period from upward extension of the disease, one even or both showing these motor phenomena, when the lesion reaches the level of the third dorsal vertebra. A farther extension upwards may implicate the phrenic nerve at the level of the fourth cervical vertebra, and in some rare cases life is further prolonged until saccharine urine and its attendant symptoms, and greater difficulty in respiration, show that the medulla oblongata has been reached. This occurs in cases in which there is no trace of bulbar paralysis.

In cases that are tolerably common, and in which, even if improvement does not take place, life is prolonged for a considerable period, the patient may walk, but at each step the legs are lifted with difficulty, almost as if they were foreign bodies. The exertion induces rapid exhaustion and tremor, and cannot long be continued.

In others, walking is an impossibility, but the legs can be moved in bed ; in others again no movement is possible except flexion of the leg, the foot, or perhaps only of the toes. It is possible also to have a complication in which paresis of the lower limbs is associated with spasm and tremor, and the symptoms depend on dorsal transverse myelitis, more or less chronic, with consecutive descending sclerotic degeneration of the lateral columns.

It is rare to meet with Hemiplegia Spinalis. I believe when it occurs it is almost invariably the result of compression, usually of a tumour, and it leads slowly but surely to paretic symptoms of the other side as well. It is associated with change in the pupil, from implication of the cilio-spinal centre. Leyden saw it in caries of the second cervical vertebra, with dislocation and twisting of the second cervical vertebra and narrowing of the canal. Infantile paralysis may be for a long time of one side

only. Very often one side is much affected and the other very slightly.

Paralysis without trophic disturbances points to lesion of the antero-lateral columns without implication of the grey matter. It is possible that the anterior roots may be involved, but this is not a common condition. In acute myelitis the inflammation affects both the anterior and lateral columns pretty equally, differing in this from chronic myelitis, in which the lateral columns may be sclerosed, the anterior remaining free. Even in myelitis following slow compression the anterior and lateral columns suffer pretty nearly together, or rather the acute myelitis that is so often somewhat suddenly superadded to the slow chronicity of gradual compression attacks both series of columns nearly simultaneously. It is a question whether lesion of the anterior grey matter can exist with only paresis or paralysis of motion without atrophy. This condition does not occur in acute myelitis, but some specimens of the subacute form of the disease show only some hyperplasia of the neuroglia without implication of the ganglionic cells. In such circumstances the paralysis exists without atrophy and is due to lesion of the fine fibres of the grey matter engaged in conduction from the brain to the motor nerves.

Thus pathology confirms the statements of physiologists that the orders of the will as to motion are transmitted mainly down the lateral columns, partly down the anterior columns, and to some extent along the anterior grey matter.

But in spinal paralysis of children and in central myelitis Burckhardt has found an acceleration of spinal conduction (motor), whilst a retardation of this conduction is seen in myelitis affecting the white substance, and he

suggests that this variation in the rapidity of conduction may be used in diagnosis.

Spastic symptoms. For the development of such phenomena there are needed two factors, a somewhat exalted reflex irritability and a paretic if not a paralytic condition of the muscles. They are symptoms of irritation. Very severe cramped movements of the lower extremities, occurring spontaneously or excited by external irritation, were considered by Brown-Sequard as the most characteristic symptom of myelitis. Some convulsion of the muscles of the lower extremities is not uncommon both at an early and at a later stage of myelitis, and especially when the reflex centre of the part is unaffected, the lesion being placed higher up in the cord. When a muscle or group of muscles is thus affected, either by way of spasm or of fibrillary tremor, some morbid trophic condition is being ushered in, and the part affected will become atrophied, the seat of the lesion then being in the anterior cornua.

Not wholly unconnected with convulsion, and yet with a marked difference, is that very peculiar fidgetiness of the lower extremities, that in rare cases is an early symptom of myelitis. The patient will sometimes state that this is induced by an itching of the skin, but more frequently it is independent of any such sensation. The patients simply cannot keep their feet still, and although the movements cannot be said to be quite involuntary they are made almost against the will of the patients, and cause them much annoyance. In one case I have seen the symptom accompanied by slight numbness of the soles of the feet and a decided shrinking from contact with a hot sponge at the tenth dorsal vertebra.

Contraction, in its more permanent form, is a late



symptom of chronic myelitis, especially of lateral sclerosis. It is not a symptom of the more severe cases of acute myelitis with rapid softening. In such cases the muscles become flabby, lax, without rigidity. In less severe cases, however, particularly where the lesion lies above the reflex centre for the lower extremities, and where, as in gradual compression of cord, the progress of the disease is slow, a slight touch on the foot may cause a tardy contraction of the leg upon the thigh, which may persist for hours and resist all attempts to overcome it. The thigh may be flexed in this way upon the trunk, the knee upon the thigh, the toes upon the sole of the foot. In one case under care, contraction both of the knees upon the thighs and the thighs upon the trunk was set up many times a day for a whole year, the contraction giving way during sleep and allowing the legs to fall back again into their normal position without any power of voluntary movement on the part of the patient. The case was one of caries of the bodies of the two upper dorsal vertebræ. When the angular curvature was hard and ankylosed this tendency to contraction gradually ceased, and the patient regained partially the power of standing and even of walking a little. I presume this occurs from a restoration of the inhibitory influence of the upper cord and brain on the lower reflex centres. Besides these reflex contractions and those due to the action of healthy antagonist muscles, whose influence masters the paralysed portion of a limb, in acute myelitis direct irritation of the white parts of the cord may induce contraction. Judging by what is known of this symptom in connection with lateral sclerosis, its occurrence in acute myelitis will point to implication of the lateral columns.

Reflex irritability is seldom diminished at the very

commencement of myelitis. It is generally exalted in the early stages, unless the lesion has gravely attacked the lumbar swelling. The reason for this is doubtless the one given above, that the lesion lying between the brain and the reflex centres for the lower extremities cuts off the latter from the inhibitory influence of the former. If the lesion does not spread extensively, this increase of the reflex irritability may persist for some time; it will recur to its normal state if the inflammation of the cord is subdued and the normal conduction through the cord restored.

On the other hand, the reflex irritability wanes, and even vanishes altogether, in those cases of myelitis in which the lesion extends downwards. Its vivacity in the early stage and its diminution later are valuable points both in deciding on the part of the cord diseased and in prognosis. Its absence is always a grave symptom, and in those cases of lumbar myelitis, in which reflex action is rendered impossible within a few days from the beginning of the illness, the prognosis is bad: it means not only myelitis of the lumbar bulb, but of the grey matter of the bulb, and is therefore accompanied with paralysis of the sphincters and its consequences and with trophic disturbances of a severe character.

In compression myelitis the increase of reflex irritability seems to depend upon some increased excitability of the grey substance.

For the production of a reflex act the centripetal path lies most usually in the posterior roots, the centrifugal in the anterior. Still sometimes a motor nerve may be centripetal and centrifugal, and so may a sensory nerve, or a motor nerve centripetal, a sensory centrifugal. There seems little reason to doubt that the factor between these



two paths is found in the ganglion cells constituting a reflex centre. This connection with ganglion cells is not yet proved anatomically. It is probable that such a centre exists for every part of the body: that every sensory nerve may be centripetal, every motor centrifugal in some reflex arc: that reflex centres are connected with each other by means of the very slender fibres in the grey matter of the cord; and that thus reflex irritation may induce complicated movements, which, in the case of reflex centres habitually associated together for certain purposes, may give rise to the idea of a voluntary act, even when the cord has been completely cut off from its connection with the brain. This radiation, however, from one reflex centre to another is in the cord invariably from below upwards.

The sensory nerve, therefore, the motor nerve and the ganglion cell must all be normal if a healthy reflex act is to take place; and besides this, the excess of action is kept in check in a healthy subject by the inhibitory influence of the brain, probably the Optic Thalami, even if every reflex centre in the cord does not exercise an inhibitory influence on those below it.

In acute myelitis the most usual cause of the loss of reflex irritability is destruction of the cells of the anterior horns. But this loss will occur after a time in cases of dorsal transverse myelitis, even when the ganglion cells in the reflex centre for the lower limbs persist. It seems as though the activity of these reflex centres in man cannot persist beyond a certain time when cut off from their connection with the brain. Weiss thinks that the reflex irritability of the cord decreases immediately after it has been separated from the brain, and that this condition lasts some time; but he bases this view on cases of

paraplegia following severe and sudden injury to the cord, and the loss of reflex irritability may have been due to shock.

The paths of conduction for these inhibitory influences from the brain probably lie in the white anterior columns, and it is on the frequency with which these columns are involved, especially in compression myelitis, that the special reflex phenomena in this disease depend.

Leaving for the moment the genito-urinary functions and the process of defœcation, so largely dependent on reflex activity, we touch upon the tendon reflexes.

The more important are the patellar tendon reflex and the ancle-clonus. The former is seen in striking the patellar tendon in a leg hanging free, and so setting up muscular spasm in the quadriceps extensor of the knee. The latter occurs on sudden dorsal flexion of the foot in certain pathological conditions of the cord. They are reflex phenomena. The patellar tendon reflex is absent in locomotor ataxy from damage to the posterior roots, or lesion at the passage of these roots to the posterior cornua; absent also in disease of the grey matter at the reflex centre for the quadriceps extensor muscle, as in acute transverse myelitis and infantile paralysis; and also in any disease that would interfere with the action of the centrifugal nerve, as lesion of the anterior roots. It is exaggerated in lateral sclerosis, in transverse myelitis above the lumbar regions, interrupting the reflex inhibitory fibres passing down from the brain.

The ancle-clonus, which Erb considers reflex through irritation of the tendon Achillis, is abnormally manifest in lateral sclerosis, and in limbs paraplegic from compression of cord, or transverse myelitis above the lumbar bulb; it is accompanied by a tetanic stiffness, followed by convulsion

of one or both legs. It also is evidence of excited reflex activity of lumbar bulb with interference with the inhibitory influence of the brain.

The more superficial reflexes, from stimulus of the skin over the scapula, the abdomen, the inner side of the thigh, the buttock, and the side of the foot, are all important in mapping out the portion of cord involved in disease. In one case noted, this superficial reflex was so intense in a case of acute spontaneous myelitis that the slightest touch on either leg excited tremors in the arms and great dyspnœa.

In estimating the value of the variation in the electrical irritability of the region lying below the lesion, several points have to be borne in mind, the seat of the lesion in the cord, the intensity of the myelitis, and more than all the question as to whether the grey matter is affected or the white only. At an early period of myelitis the electrical irritability is somewhat increased. This increase seldom persists for any long period. In destructive myelitis of the lower cord it very rapidly wanes in the paralysed limbs, and no response is given to any galvanic stimulus. In inflammation of a portion of the cord lying higher up, the normal electrical irritability may persist until a late period of the disease, and even all through in cases in which the lesion allows a gradual return to health. The higher the lesion is in the cord the more persistent is the response to electrical stimulus. The greater the intensity of the disease the more rapid and complete is the loss of the electrical irritability.

But in those cases of partial myelitis, in which the grey matter is intact and the white columns only implicated, the electrical conditions are normal, or at most scarcely altered. The grey matter is very specially the



seat for the reception and transmission of electrical stimulus, and the loss of all response to this power of stimulus is a proof that the grey matter is implicated, just as the rapidity of the loss affirms that the portion of grey matter attacked is in the lower cord. Degeneration of the grey matter is shown by the reaction of degeneration, no response to faradisation, but a ready response to galvanic current. The ganglion cells, which rule the nutrition of the joints and muscles, or other lying side by side with these, are the especial portion of the grey matter that forms the centre for the reception of galvanic stimulus, and degeneration of these cells is manifested by trophic disturbances in the affected muscles, as well as by a loss of electrical irritability; and conversely in a subacute case any improvement in the nutrition of the paralysed muscles implies an improvement in the grey substance, and is usually accompanied *pari passu* by a return of the electrical irritability.

In a large number of cases of myelitis the trophic centres are not sufficiently involved to lead to great muscular atrophy. In acute hyperplastic myelitis the waste of the muscle-groups is generally well marked. Still more is this the case in those forms of chronic myelitis differentiated as chronic anterior poliomyelitis, amyotrophic lateral sclerosis, progressive muscular atrophy and pseudohypertrophy. But in acute myelitis with softening, atrophy is not an early symptom, unless the disease has involved the grey matter of the cervical or lumbar bulb (as the case may be) primarily, or unless the softening is running a rapidly fatal course implicating the whole transverse diameter of the cord. Except where the myelitis depends on the pressure of a syphilitic gumma, or where the vessels are affected with syphilitic degeneration, an

advanced state of local emaciation of this nature is seldom recovered from. It is not however uncommon to find in a subacute case, in which the other symptoms show a tendency to improve, that a certain amount of atrophy in the muscle groups in the lower extremities will be recovered from, and the partially atrophied muscles be restored almost to their normal condition. In most cases the muscles of the lower extremities are unequally atrophied, and various deformities are produced from the influence of the healthy antagonist muscles being persistent, and being no longer counteracted by the muscles that have suffered atrophy. These deformities must be distinguished from those caused by morbid contraction of muscle.

Other trophic disturbances are amongst the most common and troublesome phenomena of a severe case of myelitis, affecting the whole thickness of the cord. Decubitus particularly, not only wherever any pressure may impede the nutrition of the skin, as on the sacrum, the trochanters, the elbows, &c., but also without this pressure where the skin may be slightly irritated as by the dribbling of urine. In somewhat rapid cases the formation of bullæ or vesicles on the back and the lower extremities, or indeed on any part below the lesion, is not uncommon; and these bullæ, when they burst, are often the starting point of fresh bedsores. Neither decubitus nor the formation of these bullæ are necessarily fatal signs. They may be recovered from. They are of use in prognosis according to the period at which they shew themselves; the earlier and the more severely they appear the more destructive is the lesion of the cord.

Muscular atrophy only occurs if the system of motor nerve cells of the anterior cornua is implicated, as in



acute central myelitis, spinal apoplexy, fracture or luxation of vertebræ pressing on the central cord, or in infantile paralysis and spinal paralysis of adults, besides the chronic lesions above-mentioned.

Trophic disturbances of the skin, bullæ, papulæ, urticaria, zona, pustular eruptions may occur in the course of the nerves which have been the seat of lightning pains. The alteration in the texture of the skin, glossiness of skin, or increased thickness of it are connected with a similar lesion. These appearances, like very acute bed sore, owe their origin not to lesion of the gangliar cells of the anterior coruna, but to destructive lesion (some authors say irritation) of the central and posterior grey matter, and perhaps mainly of the posterior white fasciculi, or to lesion of spinal ganglia. Acute bed sore is not met with except under circumstances of paralysis of sensation. Charcot records three cases of hemiparaplegia, in which from an unilateral wound of the spinal cord there resulted paralysis with hyperæsthesia on the side of injury, whilst on the opposite side the leg retained the power of motion but was absolutely anæsthetic. In these cases a bed sore formed on the buttock of the anæsthetic but non-paralysed side.

An interesting case occurred in the Bristol Infirmary under my care in 1876. A woman, aged 50, had been suffering for three weeks with motor and sensory paraplegia, paralysis of sphincters, &c. On admission she was found to have a large central bed sore. Transparent bullæ, varying in size, were developed on the knuckles of the right hand and on the right hip, travelling up the right side as high as the fifth and sixth ribs in spots where there was no pressure, also on left side of the jaw and on the lobe of the right ear. Ten days before death bullæ

appeared on the inner side of the left knee and right thigh, at the bend of the left elbow and on the left shoulder. She lived twelve days after admission. At the autopsy the grey matter was scarcely diseased except that the vessels were dilated and the connective tissue somewhat increased. The ganglionic cells were normal, but the white columns, both antero-lateral and posterior were softened, so that the nerve-tubes could not be recognised.

The trophic disturbances of joints may be either congestion of the synovial membrane from vasomotor paresis, or a sudden swelling of the joint with extreme and rapid wearing down of the articular extremities. These abnormalities are due to lesion of the anterior cornua.

The growth of bone is arrested from the same cause, especially in infantile paralysis.

Certain phenomena, believed to depend upon vasomotor influence are seen in some cases of acute myelitis.

Œdema is common, sometimes coming on *pari passu* with the paralysis, sometimes at a later period of the disease, due partly to the inactivity of the limb or to the dependent position in which it may lie, but partly at least to a paresis of the vasomotors, inducing first vascular dilatation, secondly œdema.

Variations in temperature are seen in most cases of acute myelitis, and are general and local. At an early stage the paralysed limbs shew a slight increase of temperature, but this rapidly changes, so that eventually the temperature of the paralysed limbs is about  $1^{\circ}$  C. lower than of the healthy ones. This depends on vasomotor paralysis, which at first allows an influx of blood into the parts. But this very hyperœmia of the paralysed parts affords a larger surface to the process of cooling. The vasomotor disturbance in a part is proportionate to the

degree in which its vessels are cut off from the possibility of reflex stimulus. The degree of temperature of a part depends not so much upon the amount of blood contained in it as on the amount circulating in it.

The effects of inflammatory lesion of the cord upon the general temperature of the body are somewhat more abstruse than this, and investigations by the most careful observers have seemed to lead to very varying opinions. On this question, as in so many other points in the symptomatology of the spinal cord, the cause, extent, intensity and course of the myelitis have to be considered. A spontaneous myelitis cannot be compared with myelitis arising from a severe accident, nor with compression myelitis from caries of a vertebra.

Severe injuries to the cervical cord have frequently led to extreme elevation of temperature, partly by interference with the inhibitory influence exercised by the medulla oblongata on the production of heat, partly by vasomotor paralysis. This elevation of temperature is not persistent, although in rapidly fatal cases it seems to be so. In more protracted cases it wanes, and in some, even from the first, the temperature is diminished. This early diminution is the effect of shock, of vasomotor paralysis and diminution of reflex irritability. It is remarkable that in Fischer's experiments an injury of the cervical cord produced no rise of temperature, provided the anterior columns were spared. He believes that a centre exists in the anterior portion of the cervical segment of the cord possessing a controlling or regulating power over the temperature, so that when excited the temperature of the body falls, when irritated it rises. The elevation of temperature so frequently seen after severe accidents to the cervical cord so far bears out this view, that the cord under



such lesions is intensely irritated. The slow growth of a compression myelitis, especially in the early stages, accounts (by the faintness of the irritation to the cord) for the slight elevation of temperature of the body. This centre is probably in the upper cervical cord, as Wagstaff gives a case of fracture and dislocation of the sixth cervical vertebra, with death in 48 hours, where temperatures of  $92^{\circ}\cdot3$  on admission and  $81^{\circ}\cdot75$  Fahr. forty-five hours after the injury were recorded. It is evident that lesions of the cervical cord are more frequently followed by elevation of temperature. In a case now under observation, with injury to the lower cervical cord, the evening temperature at first was  $102^{\circ}$  Fahr., and after five months is only just reaching the normal point.

But a high temperature is compatible with myelitis of the lumbar bulb. In one case of spontaneous myelitis in the Infirmary in 1873 a lesion of this nature was followed by a temperature often reaching  $102^{\circ}$  and once  $106^{\circ}$ . In another case, with myelitis from accident at twelfth dorsal vertebra, the temperature rose to  $101^{\circ}$ .

In acute myelitis of any part the general temperature will be raised in the early stage, as in any other inflammatory process in the body. We may perhaps accept the followings points to guide us:—

1. If the acute stage of myelitis be passed the temperature may rise again from the spreading of a severe bed sore, or from cystitis.

2. Acute myelitis, or sudden lesion of the upper cervical cord, and especially of the anterior columns, will more usually induce great elevations of temperature than lesion of any other portion of cord.

3. The depressing influence of shock will, however, lead to exceptions to this rule.



4. Many cases of this disease, and particularly of myelitis from compression, are examples of a fact to which the attention of the profession has been called by a high authority, that many so-called acute diseases are really acute exacerbations of chronic ailments. The very gradual growth of symptoms often in spontaneous myelitis up to a certain point shew this; and paralysis and other phenomena accompanying compression myelitis may persist and slowly increase for years, when suddenly a new series of symptoms of great intensity will supervene, causing death in a few days or weeks, and at the autopsy a recent softening will be revealed supervening on a slow chronic increase of the connective tissue. This fact, too often lost sight of, will account for great variation in thermometric records in cases of myelitis: slow chronic cases shewing a long persistence of the normal temperature, then a sudden exacerbation of symptoms and temporary rise of temperature, followed possibly by a decrease as death approaches.

5. In poliomyelitis anterior acuta, the general thermometric condition is usually one of depression, except perhaps in the earliest stage.

As an accessory to this question of temperature, the peculiarities of sweating must be noted. Leyden states that the paralysed parts may sweat much, whilst the healthy parts remain dry, but that this phenomenon may be reversed. The variation in the sweating of the paralysed and healthy parts is not a very usual symptom. When it occurs, the paralysed parts may sweat in excess in the early stages of the disease in its acute form, whilst the decrease of this function in the paralysed parts takes place at a later period, when the vasomotor paresis has caused a partial stasis in the circulation of the inert

muscles and of the skin that covers them, and all function is necessarily in abeyance.

The bladder is frequently affected in myelitis; less frequently in cases of slow compression than in cases of accident with fracture of vertebræ, even if the injury be high up in the spinal column. In acute spontaneous myelitis, the paralysis of the bladder is a very early symptom, and retention even for a long period of the disease is more common than incontinence. In polio-myelitis anterior acuta the bladder is usually unaffected.

Except in cases of severe shock, or a rapid softening affecting the lower dorsal region, the detrusor muscle is paralysed before the sphincter, owing possibly to the fact that the former derives its motor power from the second sacral nerve, the latter from the third. The sensory nerves are connected with the hypogastric plexus of the sympathetic, and the movements of the bladder are essentially reflex. The reflex centre for the bladder can be excited by irritation of the crura cerebri, the corpora striata and corpora restiformia; and for the normal function of micturition the co-operation of the brain is necessary, but not necessarily consciousness. The motor impulse is transmitted from the cerebral centres down the anterior columns to the sacral nerves that supply the bladder. For normal micturition, therefore, there is needed not only integrity of these cerebral centres, but integrity of the anterior columns, and integrity also of the lumbar bulb, from which these sacral nerves derive their origin. Lumbar myelitis therefore is the most rapid cause of paralysis of the bladder. Next in order is any sudden lesion of the anterior columns above this point: but it is surprising how diseased these columns may become, and yet bladder paralysis be delayed, if only the lesion is gradual. This bladder paralysis leads

to imperfect evacuation of urine, catarrh of bladder, and perhaps surgical kidney, uræmia or pyæmia.

Retention is the primary bladder symptom in sudden destruction of the cord, when the brain influence is cut off, and is due to shock. The detrusor muscle seems paralysed, whilst the sphincter is in a state of spasm, or the shock causes so much dulness of reflex activity in the lumbar bulb that the influence of the centripetal nerves irritated by the fulness of the bladder does not excite the reflex act of contraction of this organ sufficient to overcome the action of the sphincter. This condition rapidly gives place to incontinence. If the lumbar bulb recovers from shock, and is itself uninjured, the incontinence takes the form of occasional evacuation of the bladder, without of course any sensory consciousness on the part of the patient: just the condition in fact that obtains so often in various forms of mental disease, in which the perception of sensation is dulled. If, on the other hand, the lumbar cord is itself affected, the incontinence amounts to a constant dribbling away of urine, without at any time a complete evacuation of the bladder, and leads rapidly to cystitis and its consequences.

Paralysis of the sphincter ani is often preceded by cramp and severe pain in that muscle, with obstinate constipation: and incontinence of fæces only follows at a later period. Apart from the condition of the muscles of the anus, myelitis is sometimes accompanied by severe constipation, due partly to paresis of the abdominal muscles, but mainly to the paralysis of the hypogastric plexus. That this does not occur more frequently is probably owing to the fact that so much of the intestinal canal receives nervous influence from large branches from the aortic plexus above the seat of lesions situated low down



in the cord. Probably the more gradual appearance of fæcal incontinence is partly due to partial paralysis of the hypogastric plexus, rendering the passage of the fæces into the rectum a very slow process. During this tedious passage from the colon into the rectum, the watery portions of the fæces are absorbed, and the hardened masses that remain find, in the absence of the extrusive help from the abdominal muscles, a mechanical difficulty in passing away, even though the sphincter be paralysed. In a case lately under observation, where the patient, a young girl, had been exposed to great fatigue, and complained of acute pain in the sacral region, there was complete paralysis of the hypogastric plexus for many weeks, without any affection of the lower limbs.

A paraplegic woman will menstruate regularly, and may bear children ; the sympathetic being the motor nerve of the uterus, as Frankenhaüsen says.

In man the sexual power is not materially interfered with in slight cases. Erection is common in early stages of myelitis, and is a special symptom in injuries of the cord for hours or even days after the injury, with or without fracture of the vertebræ ; it is a symptom of irritation, and is induced by reflex excitation from the bladder, the skin of the thighs, &c.

But when myelitis is severe and the lumbar bulb or its immediate neighbourhood is involved, the sexual power wanes and vanishes. Like all other reflex phenomena the interruption of the connection of the reflex centre with the brain sooner or later leads to loss of the reflex function. Of course injury to the pudic nerve itself will induce entire loss of all such reflex activity. Dr. Henry Grace, of Kingswood, met with a peculiar case, in which the patient was crushed in the lower dorsal region by the



fall of a heavy weight upon him. One or more vertebræ were dislocated. There was complete motor and sensory paraplegia, including the genitals, paralysis of sphincter vesicæ. He gradually recovered after a year or more so as to be able to work in a pit. Sensation did not return to the penis, but the power of erection did, and he became the father of two children. During coition he felt nothing on the outside skin of the penis, but the pleasure in the act was as intense as before the accident. The sphincter vesicæ never recovered.

The sensory phenomena in myelitis are less striking than the motor, partly because the conduction of sensation to the brain takes place by means of a large portion of the cord, and therefore the lesion must destroy a considerable extent transversely of this organ to prevent all such conduction; partly that in compression myelitis the lesion usually involves the anterior portion of the cord first, and the effects of the compression may be for a long time confined to this region.

It cannot be denied that the physiology of the cord as regards the conduction of sensation is still somewhat obscure. It is tolerably certain that the sensory fibres cross at all points of the cord; and in partial myelitis from compression, if only half the cord is compressed, spinal hemiparaplegia with crossed anæsthesia is found. But the anatomical proofs of such sensory crossing are very incomplete. There may be seen a branch from the posterior roots on the outside of the grey posterior horn, which courses behind the columns of Clarke, and seems to bend towards the posterior commissure; but the poverty of the posterior commissure in nerve fibres seems to forbid the idea of a complete sensory crossing here. It is not impossible that such a crossing occurs in the anterior

commissure; but here the difficulty arises that no connection can be traced between the anterior commissure and the posterior horns.

Perhaps the whole of the sensory fibres do not cross in man. Leyden evidently considers the entire crossing along the whole length of the cord doubtful. Schiff's views are these:—

1. The posterior columns are the only sensory parts of the cord, and owe their sensibility probably only to the root-fibres passing through them.

2. The complete transverse section of the posterior columns by no means annuls the conduction of sensation.

3. It rather effects an intense hyperæsthesia.

4. The grey substance conveys sensation, both by its anterior and posterior portions. The less there is of grey substance the more protracted is the conduction of sensation.

5. The grey substance is not itself sensory, but it has the power of conducting sensation.

6. The posterior columns, which present points of transit for the sensory fibres, do not conduct them to the brain. But it is probable that the posterior columns do themselves conduct the sensation of touch to the brain.

Pain is not a prominent symptom in acute myelitis. If it occurs with any intensity, it may depend on irritation of the posterior roots of the nerves, especially by the pressure of cancer. Occipital pain is sometimes felt in myelitis of the upper cord, particularly in connection with disease of the upper cervical vertebræ.

Pain on pressure of the spinous processes will often determine the region of cancer, caries, or fracture of vertebræ, causing or about to cause myelitis by compression, but is not a symptom of myelitis *per se*.

A more common phenomenon is the tight band round the waist, with a sense of fulness, and sometimes of pressure on the bladder and rectum. This girdle-feeling may be accompanied by circular radiating pains, dull in character, most frequent in compression myelitis. Radiating pains are sometimes met with in the paralysed limbs, combined with an involuntary muscular twitching, and often preceded or accompanied by formication.

All these pains are symptoms of irritation, and therefore of an early stage of disease. At a later period they vanish and give place to a loss of sensation more or less complete.

In very localised unilateral compression, causing hemiplegia spinalis, hyperæsthesia may be met with on the side of the pressure.

Anæsthesia is sometimes co-extensive with the motor paralysis in myelitis, but there is no necessary relation. Sensation generally suffers later than motion, and its existence and extent lead to the conclusion that the posterior portion of the cord is affected. The anæsthetic parts may be cut off from their sensory centres by destructive pressure on their nerves or nerve-roots, by grave lesions of the posterior columns through which the nerve-roots reach the grey matter, and through which probably the sensation of touch is carried to the brain, or by disintegration of the grey matter which is specially concerned in the conduction of pain and temperature. Pathological facts seem to point to the posterior grey matter as being particularly engaged in this function; but the anterior grey matter has something to do with it, and in some slight degree the anterior columns.

Some observers have thought that the lateral columns are concerned in the conduction of touch, but the conser-



vation of sensation in lateral sclerosis seems somewhat opposed to this view.

Hyperæsthesia is rare, unless the inflammation of the cord is complicated with meningitis.

In sub-acute myelitis of the whole thickness of the cord, the conduction of temperature, pain and touch may be carried on, but with a certain retardation. This is the case even in acute myelitis with softening, as a small amount of sensation will be conducted up along the faintest and most impaired pathway, provided only a very few fibres remain intact.

In infantile paralysis, where the white columns are free from disease and the force of the lesion falls upon the ganglion cells of the anterior horns, the sensation of touch, pain and temperature are unaffected.

The consideration, then, of the symptoms of acute myelitis teaches us :—

1. That motor impulses are transmitted from the brain down the antero-lateral white columns, especially the latter.

2. That these antero-lateral nerve tubes are entering the grey matter of the anterior cornua all down the cord, form there connections with the ganglion cells, from which the anterior nerve-roots have their origin.

3. That the sensation of pain and probably of temperature is carried by the posterior roots through the posterior columns to the posterior cornua, thence more or less crosses all up the cord to the other side, and is carried up by the fine nerve-tubes in both the posterior and anterior grey matter, irrespective of the ganglion cells.

4. That the sensation of touch is probably conveyed to the brain in the same crossed direction in the cord, but up through the posterior columns.



5. That although pathological facts and experiments on the lower animals seem to prove this crossing of the sensory fibres, yet it cannot be proved to be universal, nor demonstrated anatomically, nor is it known whether it takes place by way of the posterior or the anterior commissure, or both.

6. That the connection of the posterior root with the anterior cornu of the same side seems proved by the sensory nerve so often being the centripetal channel in a reflex arc.

7. That the large and small cells in the anterior cornua serve for various reflex acts as the connecting link between the centripetal and centrifugal nerves, and form the centre of the arc.

8. That every portion of the cord forms such an arc.

9. That the brain (optic thalamus) exercises an inhibitory influence on the reflex irritability of the cord, and probably every segment of the cord exercises some inhibitory influence on the parts below it.

10. That the reflex centres of the cord derive more than an inhibitory influence from the brain, since interruption of their connection with the latter sooner or later leads to a loss of their irritability.

11. That the reflex centres for the genito-urinary system and the anus lie in the lumbar region of the cord; but that the hypogastric plexus affords the motor power to the uterus.

12. That in acute myelitis with softening electric irritability follows much the same rule as reflex irritability; but in acute hyperplastic myelitis the reaction of degeneration is the special electric phenomenon.

13. That the ganglion cells of the anterior cornua rule the trophic condition of the muscles and the bones.

14. That the nutrition of the skin (including the whole question of Decubitus) depends either on the spinal ganglion, or on the grey matter of the posterior cornua, or on the integrity of the posterior white columns, the trophic centre being unknown.

15. That no absolute rule can be laid down with respect to temperature in acute myelitis. The nature of the lesion, the amount of shock, &c., lead to great variations. Acute myelitis will always in the early stage be accompanied by an increase of temperature, and in the cervical cord inflammation from whatever cause (except slow compression) will be associated with a high degree of pyrexia.

16. That pyrexia, sweating, and œdema of the paralysed limbs depend on vasomotor paresis, and are early symptoms of it.

17. That this vasomotor paresis leads after a time to local sluggishness of circulation, and, as a consequence, to diminished temperature in the paralysed limbs.

18. That the vasomotor nerves pass from the medulla oblongata down the anterior columns.

19. That respiration is seldom interfered with in acute myelitis, unless the lesion extend to the origin of the Phrenic, but that it may be impeded by intense reflex irritability. This extension upwards is very rapid after accident to the cervical cord. (In one case in the Infirmary the respiration was affected on the ninth day after fracture of the body of the sixth cervical vertebra, and dislocation of a small piece of the intervertebral cartilage.)

20. That *acute* myelitis throws little light on the question of co-ordination, probably because in compression myelitis the anterior portion of the cord is generally first

attacked, and in almost all cases the grey matter suffers (motor paralysis being the consequence) before the posterior portion of the cord is implicated.

The case quoted by Erb, in which the movements of the upper extremities were powerful and completely correct, when the posterior columns of the lower half of the cervical cord were wholly destroyed, throws some doubt on the posterior columns being invariably the channels of conduction of co-ordination from the Pons, Corpora Quadrigemina, Optic Thalami and Cerebellum. In the lumbar cord there is some reason to believe that these channels lie in the middle third of the lateral columns.

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## SPINAL SCLEROSIS.

BY DR. SHINGLETON SMITH.

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J. J., æt. 37, a South Wales collier, was admitted to the Bristol Royal Infirmary on May 24th, 1878, in consequence of a paraplegic condition of eleven years duration. His previous health had been good; never had syphilis, but his friends reported that he had been a hard drinker. His family was healthy; there was no history of nerve disease.

His illness commenced with impairment of sensation and motion first in one leg and then in the other, which he attributed to constantly working in water. He continued his work for eighteen months after the first attack, and then after a little rest was able to work again, but had not done any work for six years before admission. Sensation had not been much impaired, and he had been able to walk about the house till nine months previously. He had complained at times of severe tingling pains in the legs, of shooting pains up the back, and had severe twitchings in the legs. There had been incontinence of urine for some weeks.

*P. C., May 24th.* A light complexioned man, not very much emaciated; looks older than 37, not able to walk or stand, can move his legs slightly on the bed, can bend the toes and flex feet at the ankle, but not able to draw up



the legs ; sensation in legs seems to be scarcely impaired, the muscles not atrophied, legs grow rigid at times, the arms also are stiff occasionally, no tremor noticed. The grip of the hands is feeble, but he has perfect control over the arms. Temperature of body 98·4, pulse 72, small. The heart and lung sounds healthy. Tongue red, appetite good, bowels usually constipated.

The mental faculties decidedly deficient ; this explains the scanty history of the patient's illness.

The muscular reaction to the induced current was found to be much diminished in both legs, but less so in the arms.

No impairment of vision was observed. No abnormality was found in the optic discs and retina.

He was ordered, ext. belladonna, gr.  $\frac{1}{4}$ , in water thrice a day.

*May 30th.* He had been getting rapidly worse both mentally and as regards general strength. He complained of great pain "all over," and rigidity both of arms and legs was more constantly present. There were no pyrexial symptoms. The urine dribbled away constantly and was ammoniacal. Bowels were constipated. No bed-sores had formed. Was ordered—

℞ Ext. ergotæ liq. ʒj  
Liq. opii. sed. ℥x  
Syr. aurantii ʒj  
Aq. menth. pip. ad. ʒj.

Misce. Fiat Haustus ter indies sumendus.

*June 6th.* He had not been able to move for some days ; was more helpless and generally weaker, but quite conscious till 8 a.m. this morning, when he became comatose. Rigidity of arms still present. Skin covered

with clammy perspiration. Pulse 136, small, thready. Respiration laboured and spasmodic. The right pupil widely dilated.

Death took place at 4 p.m. the same day.

The meagre account this patient gave of the onset of his illness led to inquiries from the medical man who had first seen him, and the following account was kindly given by Dr. C. B. Ball, of Blaenavon :—

“ I saw him first about five years ago, and as far as I remember was told that the first symptoms had appeared about a year previously. There was considerable wasting of the muscles, but he was able to walk with a stick, and had fair power of co-ordinating his movements. He was also able to stand with his feet together and his eyes shut. I tried him with both continuous and induced current and there was decidedly less reaction as compared with the arms. He was able to distinguish between heat and cold, but cutaneous sensation was imperfect. I treated him at the time with phosphorus and strychnia and faradization externally with apparently slight benefit. I certainly should not have diagnosed the case as one of locomotor ataxia when I was attending him. Could the affection of the posterior portion of the medulla be secondary and not the primary cause of disease ? ”

At the *autopsy* the following record was made :—

Face emaciated, eyes sunken, body generally well nourished and the muscles not atrophied, rigor mortis well marked.

The *brain* weighed 46 ounces. Nothing abnormal was noticed in the cerebrum, cerebellum, or the central ganglia. No excess of fluid was observed, and the membranes were healthy. In the fourth ventricle there was some discoloration close to the median line on both

sides, more especially on the right; and there was a greyish patch close to the corpus dentatum on the left side of medulla. The crura cerebri appeared to be greyish and soft. The optic nerves presented no abnormal appearances.

The *spinal cord* had a soft gelatinous-looking aspect throughout, particularly in its posterior aspect. The fluid was in excess, and the cord appeared to be atrophied. In section the posterior and lateral columns exhibited a condition of advanced grey degeneration, varying in extent in different regions. In the lumbar it was limited to the posterior columns, in the dorsal it was most marked in the lateral columns, in the cervical region the posterior and the lateral columns were of a grey gelatinous appearance throughout. The horns of grey matter were visible in all parts of the cord, but they appeared to be in an atrophic condition.

On examination of the cord after hardening by means of Müller's fluid and subsequently spirit, the following appearances were made out:—

The whole cord was slightly atrophied, and its cohesion much greater than normal. Its shape, however, had not assumed that characteristic of sclerosis of the posterior columns; there was no flattening of the posterior surface, and no disproportionate diminution of the antero-posterior diameter, excepting in the lumbar region where the lateral diameter was apparently increased, but still the posterior aspect even here maintained a decided convexity. The membranes did not present any abnormality, excepting that the pia mater was unnaturally adherent particularly along the posterior aspect of the lumbar region. The posterior fissure was nearly obliterated, the posterior columns being morbidly

EXPLANATION OF PLATE V.

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SECTIONS OF SPINAL CORD IN DIFFERENT REGIONS,  
FROM CASE OF J. J.

Figs. 1, 2, 3, 4, 5.—Sections through the lower part of medulla and upper cervical regions, shewing the irregular disposition of the sclerosed tissue.

Figs. 6, 7.—Middle dorsal.

Figs. 8, 9.—Upper lumbar.

Fig. 10.—Lower lumbar.

Fig. 11.—Caudal.

Figs. 12, 13.—Sections from the lumbar region, shewing irregular staining in patches, and adhesion of the posterior columns.







adherent, more particularly in the lumbar and lower dorsal region. The condition of the posterior fissure varied much in different sections ; in some it was normal, in others a vessel could be seen running down it almost to the bottom, in others bridges of tissue extended across the gap with fragments of bloodvessels visible between them, whilst in other sections the two posterior columns were completely adherent, there being no trace of any fissure remaining.

In transverse sections the appearances depicted roughly in the accompanying plate (Pl. V.) were visible. Beginning from below upwards, the caudal region was the least affected ; the grey matter did not present any abnormal appearance visible to the naked eye, and the thin posterior columns were apparently unaffected. In the lower lumbar region the grey matter was expanded laterally, the central commissure (the posterior grey portion) was increased in thickness, the posterior fissure indistinct, and the white matter of the posterior columns becoming encroached upon by an apparent extension of the grey matter backwards. A little higher the posterior columns had become much more largely implicated, and the condition was not perfectly symmetrical, one posterior column being much more affected than the other. In the upper lumbar region the disease was limited to the portion of the posterior columns on either side of the median fissure (the columns of Goll), leaving the posterior root zones unaffected.

In the middle dorsal region the posterior columns were much more healthy in appearance, the grey matter seemed rather abnormal in its outline, the posterior horns being thickened and the nerve roots indistinct, but there was a distinct white margin around its whole circumfe-

rence. At a spot only a very little higher up the sectional aspect was perfectly normal, the grey matter having the characteristic H form and the white matter its usual appearance, the principal feature being a diminution in the whole area of the cord. In the cervical region the posterior columns again were visibly implicated, the sclerosed portion being indistinguishable in colour from the grey central mass, which appeared to reach the surface along the posterior fissure. At the lower portion of the medulla the distribution of the sclerosed tissue became much more irregular, and the appearances depicted in Figs. 1, 2, 3, 4 were present.

At the upper part of the medulla and the Pons Varolii it was impossible to trace further any definite columns of morbid tissue, but the whole mass had an abnormally firm consistence, and was infiltrated uniformly with the morbid product.

*Microscopically* it was found that the nerve fibres in the sclerosed portions had undergone the usual wasting. On attempting to stain sections from various parts it was found that the staining agent was absorbed by the tissue very unequally. This was particularly the case with Aniline Blue-black. After some hours' immersion in a weak solution the sections had become stained in patches, numerous circular spots had become of a deep blue colour, the intervening portion not having become coloured at all. On noticing the section more carefully it was found that the blood-vessels seemed to act as foci round which the colour had been absorbed, each patch of coloured tissue having one or more vessels in its centre.

A marked feature in all the sections was an abundance of blood-vessels, more particularly in the grey



matter. Not only were the vessels abundant, but many of them were preternaturally large. On either side of the central canal of the cord the usual artery and vein could be seen, but each of them occupying the centre of a considerable space and surrounded by a mass of connective tissue. Sections mounted in glycerine shewed a similar ring of connective tissue surrounding all the small vessels in the substance of the cord; the capillary with its contained blood-corpuscles, having been stained with aniline, was surrounded by a transparent zone as wide as, frequently wider than, the diameter of the vessel between it and the nerve tissue around. Sections stained with Hæmatoxylin shewed numerous minute oval nuclei in the walls of the capillaries and in the larger vessels. Numerous spaces and lines were visible, with fragments of a vessel in most, but some of them not containing any portion of a vessel: these spaces were all occupied by a loose connective tissue resembling that seen around the vessels.

Another marked feature of sections stained in logwood was the multiplicity of nuclei in all parts of the tissue. Small round nuclei were scattered throughout in great abundance, and in all parts alike: they did not appear to be more numerous in the grey matter than in the white, and they were interspersed everywhere with the small oval nuclei of the capillaries.

Less numerous than these were larger rounded bodies occupying chiefly the periphery of the posterior columns. These did not come into view in unstained sections, nor were they visible in sections stained with aniline or eosine, but they were stained of a deep blue colour by the logwood, and stood out prominently amongst the small stained nuclei in consequence of the decided difference in

size between the two sets of bodies. They were scattered sparingly throughout the posterior columns, and few were seen elsewhere. Their margins were sharply defined, and their colour so deep that no internal structure could be perceived. They were all of similar size and uniformly circular. They could not be mistaken for axis cylinders, nor were they at all like any normal nerve structure. Their characters indicate their identity with the corpora amylacea of Virchow, and it is no new observation that sclerosed nerve tissue should be found to contain these amyloid bodies.

The central canal of this cord presented the usual appearances. The columnar epithelium was present, and a small-celled nuclear growth had taken place around it, as frequently happens in acute and chronic inflammatory affections of the cord.

The white commissure did not present any abnormality; the central grey commissure appeared to be considerably thickened.

The *grey matter* had undergone a varying degree of sclerosis in different parts; the nerve cells did not present any abnormal features, they were large and well developed in the lumbar region, and did not contain an excess of pigment or oil globules. The great increase in the number of vessels and the development of fibrous tissue around them were the principal abnormalities to be observed in the general grey matter.

The *white* columns were in no part quite healthy; the degree of sclerosis varied from only a slight condensation of tissue in the anterior columns up to complete obliteration of the nerve elements, and transformation into fibrous tissue in some parts of the posterior columns. A section through the posterior columns of the lumbar regions



shewed a mass of axis cylinders imbedded in thickened neuroglia, the white substance of Schwann having disappeared entirely. Where the disease was most advanced little more than a network of fibrous tissue remained. The process of condensation was not uniform throughout any particular columns; little islands of unaltered fibres could be seen in parts, elsewhere little patches of advanced sclerosis could be seen surrounded by a more healthy tissue.

*Remarks.* This case is of interest from the pathological view: its clinical history is too imperfect to enable one to deduce any conclusions from it. The appearances found in the spinal cord are those characteristic of sclerosis, and the posterior columns appear to be the parts primarily and most extensively involved. The diffusion of the lesions throughout the grey matter and into the lateral and even the anterior columns in the upper cervical region is a condition not usually noticed in cases of locomotor ataxia; hence arises the idea that the disease may have been that form of sclerosis described by Charcot as “disseminated sclerosis.” It is to be regretted that a more careful examination of the brain was not made: no morbid appearances such as would be expected with a disseminated cerebro-spinal sclerosis were seen by the naked eye, but a careful microscopic examination of the brain and cranial nerves was not instituted. The patient’s mental condition was consistent with a high degree of brain lesion; but, on the other hand, the characteristic feature of Charcot’s disease—tremor—was not at any time observed. The condition of the grey matter throughout the cord seems to indicate that this tissue may have been the one primarily affected, for, although the disease affects the posterior columns more than any

other portion of the surrounding white substance, yet it seems to affect the median columns of Goll rather than the posterior root-zones, the parts primarily affected in cases of locomotor ataxia, and in all cases the sclerosed portions are in direct connection with the grey substance. It would appear that the symptoms in the early stages of the disease were not those characteristic of locomotor ataxia, and the autopsy shews that the grey matter is more constantly and completely involved than any other portion. Both clinically and pathologically, therefore, we are led to the conclusion that this case was one of primary sclerosis of the grey matter of the spinal cord, with subsequent extension of the disease to the white columns.

#### CASE OF LOCOMOTOR ATAXIA.

W. W., æt. 42, gunsmith, married 12 years. No history of previous illness, and no hereditary predisposition was reported. He was always steady, and no history of syphilis could be elicited. His illness commenced six years before his admission. The initial symptoms were staggering in his walk, giddiness, and constantly increasing weakness; he had also gnawing pains in the legs (more especially along the shins), and soon afterwards in the fore-arms. The difficulty in walking soon increased, and he found it necessary to look at his feet whilst standing or walking. He got very nervous about himself, left off work, and went to the Mineral Water Hospital at Bath six months after the symptoms first appeared. He remained in the hospital 13 weeks, but he derived no benefit from the mineral water or other treatment. He found himself quite unable to work, and became an in-patient at the Bristol Royal Infirmary



under the care of Dr. E. L. Fox. At this time the symptoms were quite characteristic of locomotor ataxia ; he was able to walk well except when the eyes were closed, there was no wasting of the muscles and no implication of urinary organs.

Whilst a patient at the Infirmary considerable improvement was manifested under the influence of Ergot.

After this he went to a convalescent home at Bournemouth, thence to the Hospital for Paralysis and Epilepsy in London, afterwards to the Bristol General Hospital, and then to St. Bartholomew's Hospital ; but all this time the symptoms had been steadily growing worse, and he had not been able to walk for the last eight months.

*Condition on admission* to Bristol Royal Infirmary, April 27th, 1878. He was a rather emaciated, thin, spare, but wiry-looking, tall man, with a dusky tint of cheeks and lips. He was quite conscious, and gave an intelligent account of his history ; his mental faculties were unaffected, his memory good, but his speech a little jerky and tremulous. The facial muscles were subject to frequent twitchings, and his tongue was slightly tremulous when protruded. There was almost total loss of power in both legs ; he could not lift either from the bed or draw them up in the bed, but there was slight voluntary movement in the toes of both feet, and a little reflex movement in the toes followed any tickling of the soles. No voluntary movement at either knees or ankles could be effected.

In the arms there was partial loss of power ; but he was able to grasp the hand firmly, although he was not able to button his shirt, to pick up a pin, or to turn over the leaves of a book readily.

Sensation was not materially affected either in the legs

or arms. With the induced voltaic current it was found that sensibility in the arms was not affected, but in the legs there was great loss of electro-sensibility and great diminution of electro-contractility. A powerful current caused only slight movement in the toes. He complained of constant gnawing pains in the arms and down the spine ; these pains prevented sleep and were his principal trouble.

The muscles of both legs were excessively wasted, much more so than those of the upper extremities.

The appetite and digestion were not impaired ; the bowels acted without difficulty, there was no incontinence—urine was clear and free from albumen. The heart and lung sounds were normal, there was no rise in temperature, pulse was 100, small.

The sight appeared to be fairly good : he could read a newspaper without difficulty, but he stated that for two or three weeks it had not been so good as before. On examining the eyes with the ophthalmoscope it was found that the retinal vessels were small, the discs contracted, and the whole retina had a dull grey aspect ; no lymph or hemorrhages were present, and there was no cupping of the discs.

He was ordered *succus conii* in gradually increasing doses, beginning with one drachm every four hours, and this he continued till May 14th, when he was taking an ounce and a half of the *succus* every four hours ; at this time there was more power of movement in the toes and not so much pain in the arms. On May 20th the *conium* was discontinued as it seemed to have little effect in subduing the constant painful twitchings which the patient felt in the toes, and bromide of potassium was given in doses of thirty grains three times a day. His nights were

much disturbed by these constant fibrillary contractions, and accordingly cannabis indica (gr.  $\frac{1}{2}$ ) was given at bed time each night.

On the 24th extract of belladonna (gr.  $\frac{1}{2}$ ) was added to the bromide mixture, and an occasional dose of chlorodyne (℥xxx) given whenever the pains were very severe either by day or night.

On May 26th the temperature was found to be high— $102^{\circ}$  in the morning, and  $103^{\circ}$  in the evening. He complained constantly of pain in the arms as well as the legs; but he was able to feed himself, and the muscular power of arms did not seem to diminish. The sight was getting worse; he could not see the numbers of the beds across the ward, and he frequently saw double. On the day following the temperature had come down to  $99^{\circ}$ , but on May 31st it went up again to  $102^{\circ}$  in the evening. The pains were getting steadily worse, and there was no improvement in vision. The back was threatening to become sore, but still there was no incontinence; the urine was clear, generally acid and free from albumen. He made a complaint incidentally that he had no sensation in the penis, but still he always effected micturition without help and without getting wet. Constipation was always present, and was counteracted by an occasional enema and by doses of castor oil. He was now ordered liquid extract of ergot in drachm doses to be taken every four hours.

*June 4th.* Temperature was  $99^{\circ}$ ; no apparent change. He stated that he had lost the use of his arms at the shoulders. On the 7th, temperature was up to  $103^{\circ}$ , and then gradually fell to  $98^{\circ}$  on the morning of the 11th. The pain was now so intense that on June 14th hypodermic injections of morphia were commenced and were



continued morning and evening. On July 27th he was ordered the following mixture :—

℞ Ext. ergotæ liquidī, ℥j  
Succus conii, ℥ij  
Liq. morph. hydrochlor., ℥xx  
Aq. camph. ad. ℥jss.

Misce. Fiat Haustus ter indies sumendus.

He had become much more helpless, and could feed himself with difficulty, the food having been cut up. He was able to sit up (propped) in the bed, and seemed quite intelligent when not semi-narcotised with morphia. He constantly complained of twitching, particularly in the second and third toes of the left foot, and he could not bear to have them touched. Aconite liniment had no effect on the movement or on the pain associated with it; no local support from bandages or pressure gave relief. On August 28th the extract of ergot was increased to two drachms, and the hypodermic injections were continued. Attempts were made to omit the injections, but the patient always protested, and did not sleep or get any ease without them. Water was at one time injected, but he knew that the usual morphia had not been given, and begged that he might have it. Atropine was also given by injection, but it gave no ease and appeared to have no effect. The effect of the morphia gradually diminished, and the dose was slowly increased. From time to time attacks of acute gastric catarrh manifested themselves without cause; he would be unable to take food for a day, and became violently sick. These attacks recurred every two or three weeks. The ergot seemed to do little or no good; there was occasional temporary improvement, but the progress was steadily downwards for the most part.



The pains and spasmodic contractions continued with unabated severity, and the patient's general health gradually deteriorated. Towards the end of October he began to have less pain, but was excessively prostrate and helpless.

On November 1st he had been unable to take any solid food for a week, and had passed everything under him. Two nights before he was violently delirious, screaming out loudly, and fighting with his arms. The previous night he did not sleep, and was in muttering delirium. Pulse had been scarcely perceptible for several days. His face had a dusky hue from defective respiration: this had been constantly observed before as a result of the action of morphia, but now it had become persistent, although the hypodermic injections of morphia had been discontinued. The pupils were small and sensitive to light; the ophthalmoscope demonstrated the optic discs small, white, in a condition of advanced atrophy. There had been vomiting almost continually for three days, the vomit being of a bilious character. There was constant subsultus, but he did not complain so much of the painful contractions of the toes. Voluntary movement was still possible in the toes, and he could move the hands a little. The noisy delirium and sleeplessness had been kept in check by Chloral Hydrate and Bromide of Potassium, the morphia having been left off several days before.

He continued in a semi-comatose condition, capable of being partially roused, when he attempted to speak; but articulation was scarcely possible, in consequence of the general tremor of lips and tongue. The duskiness of face increased, the tongue became dry and brown with sordes, respiration became feebler, and the temperature fell.

On November 4th he was quite comatose; temperature at 11 a.m. in axilla was  $91^{\circ}$  Fahr. No pulse could be felt, and the heart sounds were very faint.

He died at ten o'clock in the evening.

*Note.* The morphia was given hypodermically at first in doses of one-third of a grain night and morning. The dose was gradually increased until the patient was getting a grain and half of morphia night and morning, with an occasional additional dose of half a grain to three-quarters during the day, when the pain was very severe. This amount was continued for several weeks without intermission.

*Post-mortem Examination.*—There was great emaciation, the muscles everywhere being greatly atrophied. A large bed-sore was observed on the left side of the sacrum.

*Spinal Cord.*—The dura mater presented no abnormality: the fluid was in excess, and the whole cord seemed small: the pia mater was congested, the veins being full and tortuous. The cord itself seemed flattened, the antero-posterior diameter being diminished. On laying open the membranes a well-marked grey translucent band was seen running the whole length of the cord corresponding to the posterior columns; in the lumbar region the band appeared to divide into two sections, each of which extended more on to the sides of the cord, leaving a whiter line in the centre. The whole of the cauda equina was grey and soft. On section the posterior aspect of the cord between the two posterior cornua was grey, translucent and tough: the whole cord was unusually resisting to the knife. The grey band extended the whole length of the cord, and was gradually lost in the medulla, being flattened out in the floor of the

fourth ventricle. The dorsal and lumbar regions seemed most affected. The lateral and anterior columns were small in size, but presented their natural white colour both on the surface and in section. The horns of grey matter were visible in all parts, and did not present any naked eye abnormality.

There was an excess of fluid within the cranium, but the lateral ventricles were not dilated. The brain weighed 49 oz.; its substance was firm.

The optic nerves were small and firm to the touch; both were flattened, and one was only half as large as the other, the left being the larger.

The heart weighed 9 oz.; the aorta contained numerous chalky concretions.

The lungs were slightly œdematous, but crepitant.

The liver weighed 2 lbs. 12 oz.; it was congested.

The kidneys weighed 9 oz.; healthy.

The muscular fibre of calf was very pale in colour; looking much like ordinary fibrous tissue.

The *cord* after being hardened in spirit presented the appearance characteristic of posterior sclerosis in a very marked manner.

The most striking feature at once perceived was that the whole area of sections of the cord was much diminished; this was noticeable in all the different regions of the cord, even the medulla oblongata appeared to have undergone a similar diminution in size; in the dorsal and lumbar regions the sectional area was not more than one half that of the normal cord.

Another manifest feature was an alteration in the shape of the organ, which had become much more oval laterally by a diminution of the antero-posterior diameter; the posterior surface was flattened, the portion between



EXPLANATION OF PLATE VI.

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Fig. 1.—*Posterior columns of J. J., shewing general sclerosis of the mass, and a wavy band of deeply stained tissue running through it, with its meshes occupied by atrophied fibres.*  
× 350 diamrs.

Fig. 2.—*Portion of medulla of J. J. Early sclerosis. Nucleus of pneumogastric (?) full of deeply stained nuclei, and containing engorged vessels, surrounded by great numbers of small cells.*  
× 100 diamrs.

Fig. 3.—*From W. W. Three shrivelled nerve cells from the anterior cornu.* × 350 diamrs.

Fig. 4.—*Posterior columns from J. J., shewing slight sclerosis, excess of nuclei, shrivelling of some nerve fibres, and great enlargement of a few. Amyloid bodies distributed throughout.*  
× 350 diamrs.

See also pp. 51, 52.



Fig. 1.



Fig. 2



Fig. 3 .



Fig. 4





the posterior roots having lost its normal convexity and become slightly concave in consequence of the wasting and contraction of the posterior columns. This posterior concavity was most marked in the cervical region where the lateral diameter was greatest; in the dorsal region the rounded form was maintained.

The posterior horns of grey matter were less divergent than in the healthy cord, in some parts were almost parallel, being only slightly convex on the side towards the middle line, curving slightly with the concavity on the outer side.

The membranes were abnormally adherent along the posterior aspect, but could be separated without tearing the tissue of the cord itself. The posterior fissure was for the most part completely obliterated; here and there slight fissures in the mass between the posterior horns were seen along the middle line, faint traces of the posterior median fissure, but the posterior columns could not be separated from each other without complete destruction of the tissue.

The blood vessels were abnormally abundant, more especially in the posterior columns; their walls were thickened, and they were surrounded by a quantity of connective tissue separating them from the nerve structures proper; even in the medulla oblongata and pons large holes were visible by the naked eye at the spots where the principal vessels run; the vessels on either side of the central canal were everywhere seen to be thus surrounded by a connective tissue growth.

On immersing sections of the cord in colouring agents it was found that the colour was absorbed very unequally, and differently with different reagents. With aniline blue the posterior columns and the horns of grey matter

EXPLANATION OF PLATE VII.

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TRANSVERSE SECTIONS OF SPINAL CORD FROM  
CASE OF W. W.

Fig. 1.—*Lower cervical.*

Figs. 2 and 3.—*Middle dorsal.* Fig. 3 shews a *peripheral zone which was stained less deeply with the logwood than the rest of the section.*

Fig. 4.—*Upper lumbar.*

*The shaded portions represent the area of sclerosis.*

Fig. 5.—*Half section of lumbar cord.*

- a. *Antero-lateral columns. Parallel arrangement of the fibrous trabeculæ, with a few honeycomb patches (b. b.)*
- c. *Posterior columns, completely replaced by an irregular areolar tissue, abundantly nucleated and containing numerous amyloid bodies (g).*
- d. *Posterior nerve root, replaced by a wide meshed network, the leading strands of which follow the normal course of the nerve fibres.*
- e. *Grey matter, only slightly stained, with numerous small nuclei and a few shrivelled cells. The vascular channels (f) are dilated and surrounded by dense fibrous tissue. The central canal (h) is occluded by a proliferative round-celled epithelial product.*
- g. *Large, deeply stained masses, probably amyloid, scattered throughout all parts of the cord.*







became the most deeply coloured, the grey matter less so than the tissue of the posterior columns; the connective tissue of the pia mater around the periphery of the cord also stained deeply; bands of connective tissue running through the lateral and anterior columns also became tinted, but there was no general staining of the white substance other than that of the posterior part of the cord.

Slow staining in logwood solution produced a blue tinting of the white substance leaving the posterior column and the grey matter unstained. On microscopic examination of the logwood-tinted specimens numerous minute nuclei were found to pervade all parts of the cord alike, and at the periphery of the posterior column larger rounded bodies were seen (*corpora amylacea*). The central canal was stained of a deep tint, and with high powers was seen to be occupied by a mass of small cells (like connective tissue nuclei) which appeared to have replaced the normal columnar epithelium. The nerve structures had undergone so advanced a degeneration that little normal nerve structure could be made out: in the antero-lateral columns, and even at the deepest part of the posterior columns numerous shrivelled axis cylinders could be seen, and in the grey matter a few small pigmented nerve cells were present.

On taking a section from the dorsal region, logwood-tinted and mounted in balsam, the following were the appearances to be made out on careful examination:—

*a.* The colouring was not uniform: not only were the grey matter and posterior columns less deeply stained than the anterior and lateral columns, but the marginal portions of the whole circumference of white matter had become less deeply tinted than the central mass (Pl. VII.,

fig. 3). The degree of transparency of the posterior columns was also greater than that of other regions. The anterior fissure presented no visible abnormality, but the posterior was obliterated by the complete adhesion of the posterior columns.

*b.* On examining with a low power of microscope the most striking feature was a great increase of the radiating fibres of the cord and a corresponding want of smoothness on the surface. Trabecular extensions from the pia mater seemed to split up the white columns into a great number of lobes and lobules, the normal neuroglia structure having become immensely thickened and much more fibrous in appearance. In the posterior columns the radiating trabeculæ were represented by a great number of small blood vessels: the structure of these columns was much more homogeneous in appearance, it appeared to contain less of the fibrous element than the lateral and anterior columns.

*c.* Another most prominent and striking abnormality seen with a low power was that the tissue presented here and there in patches of various sizes a loose spongy structure (Pl. VIII., fig. 2). Patches of this kind were seen scattered throughout the grey matter, and to a less extent the anterior and lateral columns. The meshes of the stroma appeared to be for the most part empty, but in the anterior horns of grey matter shrivelled nerve cells were seen to occupy the centre of some of these spaces: they varied much in size, some being no larger than an ordinary nerve fibre, others being three or four times the diameter of the largest nerve cells; small nuclei could be seen in the thickened stroma forming the walls of these spaces.

*d.* The absence of axis cylinders and other evidences



of nerve structure from the posterior columns was in most sections complete. In carmine-stained specimens the axis cylinders surrounded by white substance occupying the spaces in the thickened neuroglia could be well seen in all parts of the anterior and lateral columns. But in the posterior columns the whole mass presented a red granular appearance, with no very definite structure: isolated vessels could be seen scattered throughout this granular mass, and towards the deeper part of the columns near the grey commissure the carmine tint was deeper than elsewhere; no axis cylinders could be identified in the homogeneous mass. In the logwood-tinted specimen remains of axis cylinders could be seen scattered throughout the deeper part of the columns, with a loose and granular looking trabecular tissue between them. The superficial coating of neuroglia tissue which normally surrounds the cord was seen to be thickened (Pl. VIII., fig. 3), and to dip down into the substance of the columns at the position of the posterior fissure: it was here that the corpora amylacea were most abundant.

The structure of the posterior columns in parts where the diseased process had been most advanced was very remarkable. Here the whole tissue had been transformed into a reticular stroma, the meshes being formed by thick walls in diameter equal to one-half that of the enclosed spaces. The cavities in this stroma appeared to be empty; the walls of the alveoli contained a few very small nuclei.

*e.* The posterior root-zones were quite as remarkable in appearance as the posterior columns. The groups of fibres running outwards (longitudinally in transverse sections of the cord) separated easily from each other, leaving spaces traversed by connecting bands; the whole

EXPLANATION OF PLATE VIII.

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- Fig. 1.—Section of medulla oblongata from W. W. Median fissure in floor of fourth ventricle. Advanced sclerosis of the lips of the fissure, thickening of the ependyma, multiplication of nuclei, and presence of amyloid bodies.  $\times 80$  diamrs.
- Fig. 2.—Dorsal region from W. W. Patch of honey-combed fibrous tissue lying parallel to, and in contact with, one of the larger radiating trabeculæ. Similar structures were scattered irregularly throughout all parts of the cord.  $\times 200$  diamrs.
- Fig. 3.—Surface of lateral columns. Lumbar. Dense areolar strands running in from the thickened pia mater with amyloid bodies in circumferential tissue and débris of nerve fibres in deeper portions.  $\times 300$  diamrs.
- Fig. 4.—Transverse section of sclerotic network from lateral columns, with shrivelled nerve fibres in its meshes.  $\times 300$  diamrs.







formed a loose connective network containing numerous small nuclei, but having no resemblance to any nerve structure. (Pl. VII., fig. 5 *d.*)

*f.* The corpora amylacea were seen to occupy the peripheral portion of the white columns, being particularly abundant in the superficial dense layer of neuroglia covering the posterior columns and on either side of the posterior root-zones. On either side of the posterior median fissure they were most constant and most numerous. They were readily distinguished from the neuroglia nuclei and from the shrivelled axis cylinders by their size, their sharp outlines, and the depth of their colour; they were nearly uniform in size, being about as large as a red blood globule; their margins were smooth and sharp, and they were for the most part perfectly round; their colour was deep and uniform throughout. In carmine-tinted specimens these bodies could not be seen at all, nor were they visible in aniline-stained or untinted specimens.

*g.* The grey matter presented a finely fibrillated appearance, with much granular material and numerous neuroglia nuclei; with logwood it was much less deeply stained than the white antero-lateral columns, and slightly less so than the sclerosed posterior columns, the deeper portion of the posterior columns being more deeply tinted than the superficial part gave a sharp line of demarcation in colour between the posterior columns and the central grey commissure with the posterior roots; with aniline-stained sections nerve fibres could be traced running off from the nerve cells and forming a plexus in the grey substance. The neuroglia nuclei were distributed equally throughout the different portions of the grey matter. Corpora amylacea were not abundant in the

grey matter, excepting at the peripheral portion bordering on the posterior root-zones. The nerve cells of the anterior cornu were large, and presented the usual nuclei and nucleoli; other smaller nerve-cells were visible, many of them occupying spaces in the neuroglia matrix much larger than the contained cells, as if the latter had shrivelled; in the posterior cornu the cells were few and very small, scarcely distinguishable from the nuclei of the matrix. A group of large well-formed nerve cells occupied the position of Clarke's vesicular column. All the cells presented a granular appearance, which somewhat obscured the contained nuclei.

The central commissure was much thickened, the posterior grey portion presenting much epithelial proliferation within and around the central canal (Pl. VII., fig. 5 h), the anterior portion presenting a decided increase in its antero-posterior diameter.

In examining sections of the different regions of the cord, very little essential difference was noticed. In the medulla the ependyma was thickened and full of small nuclei. The superficial layer of neuroglia tissue was also very distinct and contained large numbers of the amyloid bodies. At the posterior part the fissure representing the open canal of the cord was lined by a superficial columnar layer of cells, with a small celled growth beneath; on either side of this fissure at its mouth was a patch of tissue in an advanced condition of sclerosis, having a honeycomb structure crowded with small oval nuclei (Pl. VIII., fig 1); this condition did not reach the bottom of the fissure, but it extended superficially along the floor of the fourth ventricle to a considerable distance on either side. The deeper structure of the medulla did not present any abnormality.

In the lumbar region the sclerosis was much less advanced than in the dorsal, the adhesion of posterior columns was less complete; the blood vessels were much more numerous than in the dorsal region, more particularly throughout the grey matter; the neuroglia nuclei were far more abundant, and the corpora amylacea were distributed more widely throughout the posterior columns instead of being collected together at the periphery.

The optic nerves were the only peripheral nerves carefully examined, and both were found to have undergone a decided amount of sclerosis. The one presented the appearance of an early stage (Pl. IX., fig. 2), the other manifested the highest degree of sclerosis (Pl. IX., fig. 1). The normal areolar tissue covering and trabeculæ were immensely thickened, chiefly in the peripheral portion of a transverse section of the cord; the granular remains of nerve tissue with shrivelled axis cylinders could be seen to occupy the spaces of the trabeculæ in the one, but in the other no trace of nerve structure could be made out, nothing remained but an areolar tissue network with vessels; the areolar spaces appeared transparent as if empty or occupied only by a transparent fluid. Logwood staining demonstrated the presence of numerous small nuclei in the areolar tissue of the nerves, but no amyloid bodies were observed. In the less diseased of the two nerves the original arrangement of the nerve fibres in bundles could still be traced, and within the bundles a few small neuroglia nuclei were visible amongst the wasted and granular nerve elements; but, in the other, the original arrangement in fasciculi was perfectly obliterated, and there remained a structure resembling that of the posterior columns of the cord where the sclerosis was most advanced.



EXPLANATION OF PLATE IX.

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Fig. 1.—*Left optic nerve from W. W.*

Fig. 2.—*Right optic nerve from W. W. × 30 diamrs.*

Fig. 3.—*From W. W. Section of posterior column in dorsal region. Advanced sclerosis.*

a. *Posterior nerve root, granular and nucleated.*

b. *Honeycomb structure of posterior columns, no trace of nerve tissue in interstices.*

c. *Small bloodvessel in centre of large space, with loose connective tissue around it. × 50 diamrs.*

Fig. 4.—*Portion of fig. 3 (marked d), magnified 500 diamrs., and shewing coarse trabeculæ in which are embedded a few compressed nerve fibres. The meshes of the network are empty. Small nuclei abundant.*



Fig. 1.



Fig. 2.



Fig. 3.



Fig. 4.





*Remarks.* This case was a typical one of posterior spinal sclerosis, both from the clinical and the pathological point of view. It was recognised as a case of locomotor ataxia by Dr. E. Long Fox and others very early in the patient's illness, and the gradual and slow but steady progress of the disease was traced at varying intervals up to the time of the patient's death. The pains, characteristic of the disease, were present from the commencement, and continued in a characteristic manner during the whole course of the illness; towards the end of life these pains were very intense, and life was scarcely tolerable even by the aid of constant hypodermic injections of morphia. The ataxic gait was also developed early, but this at last became obscured by the patient's becoming almost completely paraplegic as regards motion. No special eye symptoms had been mentioned, although when the optic discs were first examined they were found to have undergone a process of grey degeneration; later a condition of optic atrophy was observed, and this was coincident with a high degree of amblyopia, never amounting to complete blindness. It was noticed that the patient was able to appreciate shades of colour.

As regards the origin of the disease no definite cause could be elicited; there was no appearance of syphilis during life, and no traces of any decided syphilitic lesion were observed after death. Iodide of potassium had at no time produced any amelioration of symptoms or modified the progress of the disease.

The only drug which had any marked effect was ergot; in the earlier stages there was marked amendment in the patient's power of locomotion whilst taking this drug, but, during the later months of his life, it was powerless. Conium in large doses, four or five ounces of the fresh

juice, was absolutely inert; it did not abate the painful twitchings of the toes or give rise to any physiological symptoms. Belladonna was equally ineffectual; even when given hypodermically instead of the morphia no relief was felt, and the absence of the narcotic was detected by the patient.

The necessity for morphia in large and steadily increasing doses was quite an exceptional feature in this patient's history. The painful contractions, chiefly subjective (when the patient complained of intense pain little fibrillary or other contraction was visible), were so agonising that a constant condition of semi-narcotism was maintained for weeks together. If the morphia had been withheld the patient screamed and moaned to such an extent that, to prevent annoyance in the ward, the nurse was frequently compelled to call the House Physician in the night and at irregular times during the day in order that a hypodermic injection might be given. Dr. Burder, under whose care the patient had been whilst in the Bristol General Hospital, reports that the condition was almost unique in this respect; the pains were so intense and so constant that nothing short of the constant action of morphia made life bearable or gave any period of quietness to the ward.

*Sclerosis of nerve tissue* is a condition with which modern pathologists are perfectly familiar, nevertheless there are many points with regard to the nature of the condition and the appearances observed therein in which a unanimity of opinion does not yet prevail; accordingly the occurrence of two cases in which *post-mortem* examination disclosed the existence of sclerosis in an unquestionable degree affords an opportunity for



further investigation as to the appearances present and further inquiry as to the nature of the changes which exist.

The spinal cord of *W. W.* disclosed all the features given by Cornil and Ranvier as existing in interstitial myelitis, sclerosis, or grey degeneration of the posterior columns. These features may be briefly epitomised as follows:—

1. Grey colour and transparency.
2. Thickening and adhesion of pia mater.
3. Increase of the neuroglia nuclei in the early stages, atrophy of the same in the later stages.
4. Atrophy of the nerve elements.
5. Cementing of the posterior columns to each other by the development of a cicatricial tissue, which binds together the two bundles when normally they only are in contact.
6. Atrophy of the whole posterior columns, with approach of the posterior horns towards each other.
7. Separation of the nerve tubes from each other by intervening new tissue.
8. Loss of the white substance of Schwann and wasting of the axis cylinders.
9. Thickening of the walls of the small arteries and capillaries, with diminution of their calibre, and widening of the peri-vascular spaces.
10. Presence of amyloid corpuscles, particularly beneath the pia mater.
11. Wasting of the posterior roots of the spinal nerves.
12. Atrophy of the optic and other nerves.
13. The cells of the grey matter of cord not affected, although the disease may have extended beyond the

posterior and have invaded the cortical portions of the contiguous lateral columns.

The case *W. W.* may be taken as the type of the most advanced form of sclerosis. Here all the appearances mentioned by Cornil and Ranvier existed in the most extreme degree, and even more than are mentioned by those observers, for no part of the area of transverse sections in the lumbar and dorsal regions presented the appearance of healthy nerve tissue. Even in the anterior columns the nerve tubes were separated by abnormal new growth, and the grey matter presented a fibrous appearance, although the cells had not undergone an atrophic shrinking. The posterior columns were simply a mass of cicatricial tissue, in which the few wasted remains of the axis cylinders could be detected, together with a small number of very small shrivelled neuroglia nuclei.

The other case, *J. J.*, shows the appearances of an earlier stage, when the neuroglia elements are in excess, the blood-vessels abundant, and the cicatricial contraction only commencing.

A consideration of the appearances present in these two cases appears to support the view advocated by Cornil and Ranvier that a sclerosis of the cord is a chronic interstitial myelitis, and that it is an analogous condition to a chronic congestive cirrhosis of the liver, kidney, or lung. It may be said that these latter cases are scarcely analogous, inasmuch as they generally result from a mechanical congestion dependant on some previous disease elsewhere; but all pathologists of large experience have been acquainted with cases of cirrhosis of liver or kidney which cannot be traced to any previous mechanical cause, and which do not depend on either of the common antecedents to cirrhosis in these two organs, viz., spirit-

drinking or syphilis. If we exclude the cases of cirrhosis which depend on chronic hepatitis from heart and lung diseases, and those depending on alcoholic causes, the number remaining will be very few, and these are the cases analogous to those of sclerosis of the spinal cord, which are extremely rare, so much so that they have been distinguished from the ordinary form of myelitis only during the last few years.

Again, just as a slight degree of cirrhosis of liver, kidney, or lung is one of the most common conditions found in a great variety of diseases, so a slight amount of sclerosis of nerve tissue is found in a great variety of nerve diseases. These have been enumerated by Mr. Kesteven (*Brit. and For. Med. Chi. Review*, July, 1874) and need not be recapitulated.

The common factor present in all the cases where cirrhosis has occurred seems to be an inflammatory process chronic in its character, leading first to the production of new material by proliferation of the nuclei of the cellular elements of the part involved, and subsequently contraction resembling the ordinary process of cicatrisation; this contraction being the immediate cause of the wasting of the parenchymatous elements of the organ and its subsequent diminution in size and weight.

Just in a similar way it would appear that a sclerosed condition of nerve tissue may result from a great variety of conditions, all of which may be presumed each in itself to be sufficient to account for either a local or general inflammation of the tissue affected; the one common factor existing in all these different conditions is the inflammatory process, and a different result ensues with different degrees of the inflammation; in the one the cord will undergo a general softening, or an abscess may be



formed in either column, the whole texture of the cord may be disintegrated, whilst in another a slow process of connective tissue growth and cicatrisation may finally result in the condition recognised as grey degeneration.

Analogy thus indicates that the condition, sclerosis of the spinal cord, as seen in its most typical condition after death from tabes dorsalis, or as seen in the disseminated form in cases so fully described by Charcot (*Clinical Lectures on Dis. of Nervous System, Sydenham Society*), is the result of an inflammatory process. What are the facts which support this view?

The pathological phenomena which indicate its inflammatory nature are the following:—

1. The great increase in the neuroglia nuclei; this is more particularly the case in the early stages; when the disease has become advanced many of these nuclei have become so wasted as to be no longer visible.

2. The accumulation of leucocytes within the lymphatic sheaths of the vessels, and the subsequent thickening of the walls of the vessels with increase of connective tissue immediately around.

3. The great increase in the vascularity of the parts affected when in the early stages; this increase was very well marked in the case of J. J.

4. The changes in the pia mater; these are thickening, cloudiness approaching to opacity, and adhesion; similar changes result from any chronic irritative condition of the cord depending on tumour or other morbid condition.

5. The atrophic condition of the nerve elements, the disappearance of the medullary sheath of the fibres, the wasting and separation of the axis cylinders from each other by interstitial growth, are just such conditions as



would arise from the presence of an inflammatory exudation and its subsequent cicatrisation. The ultimate condition of the posterior columns from a case of tabes resembles that of an organised mass of inflammatory exudation.

6. The occurrence of the corpora amylacea, their constancy in all cases of localised and even of disseminated sclerosis, and the constancy with which they are found in the ependyma of the ventricles in cases of chronic meningitis.

The clinical history of cases of posterior sclerosis also affords abundant evidence of the inflammatory nature of the change.

The following points may be selected:—

1. The mode of onset, which not unfrequently resembles that of myelitis, and may be traced to exposure to cold and wet.

2. The occurrence of pains, generally the first symptom mentioned. The lightning pains of “ataxia” are amongst the most characteristic of its symptoms, and are at least suggestive of a sub-inflammatory condition of the membranes of the cord. The severe pain and the spasmodic contractions of acute meningitis are sufficiently constant to indicate the probable nature of the “rheumatic” pains of tabes dorsalis.

3. The occurrence of fibrillary muscular contractions point to an irritative condition of the nerve centres in the cord such as would result from a congestive state.

4. The existence of gastric crises, which are by no means of rare occurrence, may be accounted for by a temporary increase in the inflammatory mischief, occasional exacerbations of the process. The rise in temperature and the occasional attacks of vomiting can

scarcely be accounted for otherwise. The patient W. W. had frequent attacks of this kind, which were clearly inflammatory in their nature, in which the temperature of the body rose to 103° Fahr.

5. The effect of drugs—in the early stages of the disease treatment adapted to myelitis seems to be most effective—Ergot, presumably by constricting the vessels, and therefore diminishing the blood supply, is found to be of temporary benefit.

An examination of the two cords now described almost of necessity raises the question, What is the nature of the amyloid bodies (so called) which are so abundantly present? That their name given by Virchow does not express their real nature there can be little doubt. Wagner says of them:—

“Corpora amylacea are commonly ranked with lardaceous degeneration. They are probably also nitrogenous.”

That they are protoplasmic in nature seems to be indicated by the readiness with which they take up colouring matters, particularly Hæmatoxylin.

Their size precludes the supposition that they can be nuclei; their smooth margins and homogeneous texture point to the same conclusion.

They appear to be connected with the neuroglia tissue rather than the proper nerve elements, and accordingly exist in abundance wherever the neuroglia has undergone a chronic inflammatory change.

# THE PATHOLOGY AND TREATMENT OF CHRONIC OSTEO-ARTHRITIS.

BY MR. GREIG SMITH.

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FEW subjects have received more attention at the hands of surgeons and pathologists than chronic disease of the joints. But much still remains to be done. We are more familiar with the gross lesions which, in their finality, constitute the disease called chronic arthritis, than with the natural history of the pathological processes which lead up to it. Our treatment is on the same level as our pathology. We step in only after the disease has committed its ravages, and are content if we save the limb, seldom or never being able to preserve the joint. Clear notions of the first departures from the healthy state, and of the course of the morbid processes, are necessary to guide us in our schemes for cure; and close and careful clinical observation is wanted to increase the stock of symptoms which will lead to a more exact knowledge of the stage of the underlying disease.

In the end the lesions characterising chronic diseases of the joints are very much alike. But, in the beginning, and for a considerable period in their course, there is sufficient variety to constitute at least two distinct species of disease. One commences in the synovial membrane, the other in the cancellated ends of the bones. Up to a



does not describe, probably because the acids which he used in making his preparations had done away with its appearance. He speaks of a "zone of altered cartilage" which he found in this situation; and this is probably the zone I speak of, deprived of its calcareous salts.

In properly made sections, it is easy enough to make out a calcification of the cartilaginous matrix, precisely similar to what is observed on a much larger scale in the ordinary process of ossification. Yet no one seems to have accurately described its appearance or explained its real nature; though many have given descriptions of some structure occupying the position of this zone of calcification. As its history is interesting I shall shortly relate it.

In 1841 Toynbee\* described an "articular lamella" underlying articular cartilage and completely cutting off its connection with the bone. He considered it impermeable because he had never been able to discover orifices in it, nor to force mercury through it. It was granular, highly refracting, contained no true bone cells, and had, scattered through its substance, small groups of cells not unlike those of cartilage.

In 1853 Tomes and De Morgan† gave a fair description of this structure under the name of "ossified cartilage"; but, so far were they from recognising its physiological harmony with the ossifying process, that they looked upon it as an altogether abnormal structure in the economy, and accounted for its existence as a special and exceptional adaptation to function. About this time, Sharpey imperfectly described it, but gave no hint as to its true meaning.

In 1859 Barwell‡ described this same "articular

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In 1860 Hulke\* described this lamella, or tissue which lies between articular cartilage and bone, as a "stratum of cartilage impregnated with earthy matter," and denied the existence of tubules. This account, so far as it goes, I believe to be correct; but the true meaning of the structure does not seem to have been grasped.

The structure in question I believe to be the true homologue of the calcification of matrix which precedes the development of bone in cartilage. In no specimen of articular cartilage have I found it entirely absent. It may be described as a layer of cartilage, impregnated with nodular deposits of lime salts, extending from the line of true bone a varying distance up the articular cartilage, and enclosing groups of rounded cartilage cells. The deposit varies in coarseness from a ground-glass appearance up to a rough mosaic, the individual nodules being sometimes nearly as large as red blood

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process is essentially the same ; the only differences are as to the magnitude of the ossifying area, and the rapidity of the process.

Many peculiarities are to be observed in different articulations, and at different points in the same articulation, particularly where the cartilage merges into the capsule. But, as I here refer to the normal process only, with the view of elucidating the diseased state, the above sketch may suffice. Having made out roughly the double function of articular cartilage to produce new bone on the one side and new cartilage on the other, with the double end of making good loss from compression and from friction ; and understanding the general scheme whereby these ends are attained, we are in a position to grasp the sequence of events in disease. It will be seen that the pathological states are readily grouped as perversions of the physiological changes. Though many conditions will have to be described where the departures from the healthy state are striking and often apparently divergent, it will appear that there obtains a very complete similarity through the history of the healthy and the diseased states.

The *Pink Marrow* in the cancellated ends of long bones demands some consideration. A highly vascular and vitally sensitive structure, closely packed in the bony framework, it must play a very important part in diseases affecting this part of the organism. It carries with it an amount of nutriment apparently far in excess of the demands of the bony tissue which it supplies. We see that the vitality of ordinary compact bone is maintained by a blood supply many times less than is afforded to the scattered trabeculæ in the ends of the long bones and in the short and flat bones. What is the destiny of this surplus nutriment in the cancelli ?



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Many peculiarities are to be observed in different articulations, and at different points in the same articulation, particularly where the cartilage merges into the capsule. But, as I here refer to the normal process only, with the view of elucidating the diseased state, the above sketch may suffice. Having made out roughly the double function of articular cartilage to produce new bone on the one side and new cartilage on the other, with the double end of making good loss from compression and from friction; and understanding the general scheme whereby these ends are attained, we are in a position to grasp the sequence of events in disease. It will be seen that the pathological states are readily grouped as perversions of the physiological changes. Though many conditions will have to be described where the departures from the healthy state are striking and often apparently divergent, it will appear that there obtains a very complete similarity through the history of the healthy and the diseased states.

The *Pink Marrow* in the cancellated ends of long bones demands some consideration. A highly vascular and vitally sensitive structure, closely packed in the bony framework, it must play a very important part in diseases affecting this part of the organism. It carries with it an amount of nutriment apparently far in excess of the demands of the bony tissue which it supplies. We see that the vitality of ordinary compact bone is maintained by a blood supply many times less than is afforded to the scattered trabeculæ in the ends of the long bones and in the short and flat bones. What is the destiny of this surplus nutriment in the cancelli?

Pink marrow is composed of an abundance of round lymphoid and other cells, lying in a fine fibrous reticulum freely traversed by blood vessels. The great mass of cells are scarcely distinguishable from white blood cells. They are rounded, contain granular matter and a few nuclei, and exhibit amœboid movements. Many cells contain fat globules; and, as we approach the yellow marrow, we meet, in increasing numbers, large spherical cells distended with pure fat. The supporting reticulum is indistinguishable from that in the pulp of the spleen and lymph glands. The vessels have very thin walls, most are destitute of true adventitia, and are immediately surrounded by the tissue proper of the medulla. But the most suggestive structure found here is a large round cell, filled with granular contents of a brown colour, and shewing occasionally the outlines of a few red blood cells in some part of its substance. They are in fact the counterparts of the compound granular cells found in the spleen, which are known to be intimately connected with the breaking up of the old, and the manufacture of the new, red corpuscles of the blood. There can be no doubt as to the structural similarity of these cells in the spleen and in the marrow; it still remains to be shewn that they are functionally identical. Some time ago Newman and Bizzozero described the transformation of the lymphoid cells of the marrow into the red corpuscles of the blood; and quite recently this opinion has been confirmed by further experiments of Bizzozero and Salvioli on guinea pigs and dogs.

Thus, in the red marrow, we have all the essentials in the structure of a vascular ductless gland, and we find present bodies which suggest functions similar to those of a blood forming organ. There is, therefore, a strong pre-



blood-vessels, in fact, with a concomitant diminution of vascularity, I should infer that their chief source was by proliferation. The reticulum in which these cells lie, becomes obscured with a granular protoplasmic product holding numbers of irregularly formed nuclei in its substance. The walls of the vessels are clogged with similar protoplasmic material, to which adhere groups of young round cells. Numerous friable fibrinous threads everywhere traverse the inflammatory tissue.

Soon the young cells exhibit signs of decay. Their walls break down, and their contents commingle with the swollen areolar tissue and protoplasmic material. By teasing and brushing, protoplasmic masses containing nuclei and shrivelled and broken down cells are met with very similar to giant cells. In many specimens, however, no such cohesion of elements is seen, and the slightest touch is sufficient to set the whole granular mass floating in finely divided particles. There is always a varying number of the round granular lymph-like cells.

How frequently chronic inflammations of the marrow undergo complete resolution it is impossible to say, for they do not appear on the *post-mortem* table, and it is very rarely, and then with much uncertainty, that we can infer their existence during life. Indeed, it is probable that the products of inflammation just described are already on their road to passive decay. If examples of healthy resolution of inflammatory products in the disease under consideration be rare, the same cannot be said of unhealthy forms of metamorphosis. Caseation, as a result of strumous inflammation of marrow, is exceedingly common. Caseous foci of all sizes from a millet seed to a filbert nut are found in varying numbers in most specimens. Sometimes these masses are surrounded by a well formed



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capsule of firm fibrous tissue, closely adherent to the bone. Such encapsuled masses consist of curdy pus, white creamy matter, or dense caseous substance. The smallest of these caseating foci are very similar in appearance to miliary tubercles; and, by those who do not believe in the more modern theories of tuberculosis, would be described as such. It is possible that such small nodules may truly arise from a local infection from caseated matter; just as we find groups of undoubted miliary nodules around a caseated broncho-pneumonic patch in the lung. I believe, however, that they can rarely, with propriety, be described as true tubercle of bone or of marrow, but that they are simple degenerations of inflammatory products.

In the substance of the degenerate mass of purulent or cheesy material there is seldom visible the slightest vestige of bone. This is all the more curious that, in macerated preparations, we sometimes see, in a cavity, a loose piece of cancellated tissue completely cast off from the surrounding bone. In the process of caseation either there must be very rapid and thorough breaking up of true bony tissue; or the caseating centre must, as it increases in size, cause absorption or breaking up of the bone at its periphery. The loose necrotic pieces met with in macerated specimens had then, in all probability, been set free by more active tissue changes than occur in the caseating process.

An equally common and frequently concomitant form of degeneration is the fatty. Specimens obtained from operations for excisions of joints, shew this condition more frequently perhaps than any other. The healthy pink marrow is replaced by an oily diffuent tissue of a pale yellow or light brown colour. Examined minutely, it is found to consist of fatty globules and granules;



EXPLANATION OF PLATE X.

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## DISINTEGRATION OF BONE IN OSTEO-ARTHRITIS.

Fig. 1.—*From the diseased astragalus of an adult. The bone is breaking up into irregularly shaped pieces. The bone corpuscles are passive.*  
× 300 diamrs.

Fig. 2.—*From the head of a femur in morbus coxæ. The ordinary form of atrophic caries. The bony matrix is everywhere granular, the lacunar margins are irregularly corroded, and the bone corpuscles are stunted and shrivelled. The bony tissue breaks up at once into minute nodules.* × 200 diamrs.



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Fig. 1.



Fig. 2







passive crumbling necrosis than active physiological existence. In tubercle, and similar structures, where there are crowded cells with dim contents, a little pressure on the glass cover, to make a rather thick section more translucent, will often, by causing a fusion of the protoplasmic debris, produce an appearance exactly similar to that just described. They are giant masses of granular protoplasm, but they are not active giant cells.

Caries takes place as a passive falling to pieces of the bony tissue lying in contact with the granulation material, and surrounding the bone cells and Haversian spaces and canals (Pl. X., fig 2). The bony detritus, in a state of fine sub-division, commingles with the surrounding degenerate products of inflammation. The free margins of bone are corroded and irregular in outline, and their structure is rough and granular. The lacunæ enlarge, and their boundaries become irregular, granular and corroded. The canaliculi are increased in calibre, and present the same roughened walls as the lacunæ. The contents of both lacunæ and canaliculi are choked with granular detritus. Frequently the canaliculi and lacunæ attain to large dimensions, and open into each other, forming irregularly shaped canals in the bone substance, as large as ordinary Haversian canals. Such canals usually run at right angles to the long axis of a bony spicule, and are found in greatest abundance where the spicules are thinnest. They are, in fact, evidence of close proximity or abundant supply of nutritive pabulum; and their course is the shortest route between the sources of nutrition. There is wonderfully little evidence of proliferation of bone cells inside these canals; and I have never met with a blood vessel in their interior. This is the canaliculization of bone tissue described by Volkmann, and is the

EXPLANATION OF PLATE XI.

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Fig. 1.—Osteoblasts. Section from the cancellous bone in the upper extremity of an infant's tibia.  
× 300 diamrs.

- a. *Lacunæ, bone corpuscles, and canaliculi.*
- b. *Fusiform and club-shaped granular bodies closely set and large where farthest from the formed bone; smaller and less thickly laid down in contiguity with the bone.*
- c. *Free nuclei, deeply stained.*
- d. *Transformation of nuclei into bone-corpuscles. Here the fusiform osteoblasts (so-called) are lost sight of.*

Fig. 2.—Peculiar disintegration of bone, probably a separation of the original osteoblastic deposits. Femur in morbus coxæ. × 350 diamrs.

- a. *Bone corpuscles enlarged or subdividing.*
- b. *Breaking down of lacunar walls, and liberation of bone cells.*
- c. *Club-shaped pieces of disintegrating bone.*
- d. *Granular debris, free nuclei, fusiform bodies and fibres.*

Fig. 1.



Fig. 2







result, not of absorption by advancing marrow, but of peripheric disintegration around the spaces in healthy bone. After the canals have been formed the marrow creeps up and fills them, but the marrow is not primarily concerned in their production.

The disintegration of bone does not always leave finely molecular products alone. Bony particles, as large as lacunæ, or even larger, are sometimes set free in the granulation tissue (Pl. X., fig. 1). In some instances these particles are cast off with a regularity of size, and a similarity of form which suggest the influence of more than casual conditions (Pl. XI., fig. 2). At the bony margin club-shaped pieces of fairly homogeneous bone, with clearly defined outlines, are seen clinging to the parent mass, or lying free in the marrow. The prevailing shape is ovoid or clubbed, but some are regularly oval, spindle-shaped or rounded. Among these, easily recognised, by their different refrangibility, are unusual numbers of healthy looking, rounded, occasionally nucleated cells. At a little distance from the bone the club-shaped bony masses are lost sight of, having evidently undergone complete disintegration; but the cells remain longer and multiply. A few short fibres are always present. Sometimes the breaking up takes place, not at right angles to, but parallel with the general direction of the bone. In this case it is possible to recognise a separation or fracture of the original bony lamellæ. This last form of disintegration, though probably owning a similar cause to the preceding, is still not the same.

The breaking up of bone into club-shaped pieces is difficult of explanation. I am inclined to account for it by reference to the original bony development through osteoblastic cells. In the process of ossification, the true

bone is first laid down, in the primary medullary spaces, as a non-laminated, faintly granular substance, by small round cells, which are derived either by proliferation from the cartilage cells, or by continuity from the underlying marrow. Lower down, the bone is deposited in concentric lamellæ around vessels. Here we meet, on the free surface of the bone, epithelial looking cells—the osteoblasts proper of Gegenbaur (Pl. XI., fig. 1). What is the peculiar function of these cells, and what determines their occasional but not universal appearance, need not be here discussed. It is not unreasonable to suppose that they absorb, or secrete in their substance, calcareous salts, and that the formed bone is their cell-contents. The bone would have different density at different portions of the cells, being most nodular at the periphery and most compact in the centre. Hence, the disintegrating process following these lines of original formation, arises the peculiar shape of the bony masses, corresponding to the shape of the original osteoblasts.

Such are the bony changes observed in scrofulous inflammation of the pink marrow. The inflammation of marrow is first in order of time as in importance; the osteoid changes are, in every sense, subordinate. The origin, maintenance, and spread of the disease are wholly dependent on the condition of the marrow; and quite independent of the passive implication of the bone. To call the general condition of the articular end caries is a misnomer in the face of the more important osteo-myelitis, which is not only the determining but also the continuing cause of the mischief.

While the marrow and bone are thus progressing to decay and death the surrounding structures are under-

going changes, hyperplastic and atrophic, which have important bearings on the issue of the disease.

The *Periosteum* nearly always participates to some extent in the inflammation. At first it suffers an augmentation of its normal function,—production of bone. Young layers of periosteal bone, not usually in great numbers, are laid down around the myelitic centre. These more recent deposits can usually be distinguished from the older bone by their irregular, somewhat areolar, disposition, and by the deeper staining they take on with logwood or aniline blue. This general increase of thickness of the compact layer is only temporary. As the disease advances the underlying old bone is thinned out from the inside; and in the end, the compact layer is even thinner than normal. In some instances, particularly in the small bones of the hands and feet, so thin does the compact shell become that it is pushed out and expanded, by the excentric proliferation of the marrow, giving rise to the condition known as “*spina ventosa*.” The ultimate result of such a condition is bursting of the bony shell and the formation of abscesses and sinuses. Such results rarely occur in the articular ends of the long bones; there the proliferating marrow usually finds its way into the joints. In the short and the square bones, where the compact shell is thinner, and where pink marrow occupies the whole of their interior, such results are more common than perforation of the articular ends.

Occasionally, in addition to true bone, masses of calcareous salts are thrown down by the periosteum. Many macerated specimens of this form of disease are covered with a layer, more or less thick, of pure calcareous material. A preparation of the lower end of a femur in the Bristol Infirmary Museum is coated with a layer of nodular



calcified material which, at some parts, is nearly quarter of an inch thick. What determines this result I am unable to say ; certainly it is of no very infrequent occurrence.

The increased formation of bone by the periosteum is essentially a conservative effort, and ought to have an important bearing on any schemes for cure. As the compact outer shell is invaded and gradually eaten up by the spreading disease, the periosteum continues to form new bone in advance ; and, if it were possible to completely remove the diseased marrow, this periosteal formation might be competent to maintain the strength and continuity of the bone.

*Articular Cartilage.*—The naked-eye appearances of an articular surface that has become involved in osteo-arthritis are, in the aggregate, distinct from those seen in a case of pulpy synovitis. In the one, the cartilage is primarily invaded from its free surface ; in the other, from its bony attachments. In pulpy synovitis the inroads are chiefly from periphery ; in osteo-arthritis, if there be any predilection, it is for the centre of the articulation. Gradual eating up by advancing and overspreading granulation tissue which frequently becomes fibrous and merges into a similar transformation of cartilage, is the rule in the synovial form of disease. Invasion from below, elevation in blisters and perforation or isolation of the cartilaginous incrustment by proliferating marrow, is the common result in the bony form. A joint moderately advanced in scrofulous synovitis would probably exhibit the following appearances. The capsule enormously thickened, and formed of lowly organised fibrous tissue ; pulpy, anaemic granulations hanging in lobes or papillæ from the interior ; the whole joint cavity much diminished,



and perhaps subdivided ; the articular cartilage rough, irregular, thickened and fibrous at some parts, eroded and perhaps completely removed at others, where granulation lobes have been lying, but, on the whole, firmly adherent to the underlying bone. In osteo-arthritis the leading changes are different. The capsule may be thickened, but seldom is it much so ; the synovial membrane tends rather to suppuration than to the formation of granulation tissue ; the joint cavity is not diminished, on the contrary, it is frequently enlarged ; the articular cartilage has lost its glistening appearance and is white, granular and opaque on the surface, but it seldom is overrun by fibrous tissue ; perforations, which may exist, are made from below, and have sharp overhanging edges, and the whole cartilage is either loosely adherent to the bone, or flapping free in the general cavity. Thus far the diseases are evidently distinct. But, towards the end, when abscesses have formed, and the whole of the tissues have undergone extensive necrotic changes, there is little beyond the excess of fibrous tissue in the synovial form to help us to distinguish the two.

The articular surface of a joint affected with arthritic caries usually exhibits the following features:—In the earlier stages, when the proliferating marrow has come close to, but has not actually invaded its substance, the cartilage is seen to have lost its lustre and polish, and to have become of a dead grey or straw colour. As the disease advances, the cartilage becomes slightly elevated over areas of different size, and the surface presents alternating undulations and depressions. The elevations feel boggy and semi-elastic on pressure ; they indicate that the cartilage is being stripped from the bone by the fungating marrow. Soon the appearance of small pinkish

spots on the cartilaginous blister, indicate that perforations are about to take place. Small holes appear with thin, shelving, ragged edges; and papillæ of granulation tissue protrude from them. The holes increase in size, while the articular cartilage continues to be elevated; and ultimately, by fusion of these perforations, flaps and islands of cartilaginous tissue are completely set free from their bony attachment. Sometimes the periphery of a blister is eaten through, and button-shaped pieces of cartilage lie in the centre of a cup-shaped mass of granulation tissue. Or the perforation may be around only one half of the blister, and then a flap of cartilage is left attached by a hinge of its substance. In a preparation of the head of the femur which I possess, the whole of the cartilage can be removed like a cap.

It frequently happens, in cases of this disease, that not all the bones entering into an articulation are affected. Thus the femur may be much diseased, while the tibia is only slightly so, and the patella not at all. But when the cartilage encrusting one bone has been perforated, the contiguous cartilages of the others soon become affected. A specimen before me shews, on the front of the outer condyle of a femur, a granulation area as large as a shilling, surrounded and partially overlapped by a thin cartilaginous margin. Corresponding to this area, the opposing patellar cartilage has been eaten away. But the erosion had been from above and not from below; and the remaining cartilage is still closely adherent to the bone. The myeloid tissue of the patella had not yet been invaded. In disease of long standing, however, all the tissues entering into an articulation become affected in the same manner and in like degrees; and when, in such cases, the joint has ap-

peared for examination, it is often impossible to say where the disease commenced.

An exception to the foregoing description ought to be made in the cases of the vertebræ and the bones of the hands and feet. In these the cartilage is often slightly and sometimes not at all affected. This arises from the fact that the fungating marrow bursts through the thin compact shell of these bones more easily than through their articular surfaces, and thus finds a vent before it has destroyed the articulation. Often however the articulation is early destroyed; in advanced disease it is always so. Still it is surprising to what an extent a phalanx may be diseased, while its immediate neighbours remain unaffected.

The minute changes in articular cartilage are varied and interesting. The histological characters vary according to the age of the patient, the stage of the disease, and the acuteness of the processes at work. Various forms of degeneration attack tissues which have suffered in different ways and degrees from inflammatory action. The histological changes are often distinct at different points of the articulation. In the midst of all this variety, it is difficult to select leading features on which to ground a generalised description. I shall therefore endeavour to group the changes under the tissue elements and the physiological functions which they may separately discharge. This plan involves a certain amount of repetition, and detracts from any possible unity and consecutiveness which a description following the course of the disease might possess; but, for a clear understanding of the whole of the results, it is, I believe, preferable.

I shall, in the first place, notice abnormalities I have found in the calcification and ossification of articular car-



## EXPLANATION OF PLATE XII.

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*Slight exaggeration of the normal process of calcification and ossification in articular cartilage. From the diseased olecranon ulnæ of a child aged 10.*

*× 350 diamrs.*

- a. *Normal cartilage cells and capsules in subdivision tending to become isolated. (a').*
- b. *Deposition of nodular calcareous material around the capsules.*
- c. *Partial solution of calcareous material with proliferation of cartilage cells.*
- d. *Further solution of salts and proliferation of cells, with the formation of true bone on the side farthest from the joint surface.*
- e. *Same as d, more advanced.*
- f. *Formed bone.*
- g. *Marrow.*
- h. *A bone corpuscle becoming embedded in the young osteoid tissue.*







tilage, and then consider in detail the various forms of degeneration and absorption which I have met with.

*Changes in Calcification and Ossification of Articular Cartilage.*—We have seen that a layer of calcification lies in front of the ossifying line in articular cartilage. This layer is much thicker in young bones than in old; its average thickness in subjects who would be most liable to articular disease is about  $\frac{1}{50}$  in. In disease there are changes in the thickness of the calcification area, and in the form and arrangement of the deposits.

Simple increase or diminution of thickness in the calcifying zone follows, as nearly as possible, changes in the blood supply. At first, with increased vascularity, there is an acceleration of the normal physiological process (Pl. XII.). The granulation tissue, as yet fairly well nourished, will have appeared at the ossifying margin, and bears with it a more near and abundant supply of plasma than did the healthy marrow. We may call it inflammation. The first effect of this inflammation in the calcifying zone is an abundant, irregular, and haphazard deposition of granular and nodular salts. The salts are thrown down everywhere in the ossifying area,—around the cells, in the matrix, on the bony trabeculæ (Pl. XIII., fig. 2), and even in the marrow substance. Surrounding the cell capsules the deposit is patchy and irregular, and rough nodules encroach on the contained cells. In the matrix generally the nodules are laid down unevenly, and there is not the same partiality for pericapsular localities as in health. A disregard for its proper nidus is an important feature in the disturbed calcifying process. Thus, rounded lumps of unmistakeably calcareous character are found lying in beds and hollows among the bony trabeculæ next the cartilage (Pl. XIII., fig. 2).

EXPLANATION OF PLATE XIII.

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Fig. 1.—*Nodular calcification at the junction of cartilage and bone in the diseased lower extremity of the tibia of a man aged 40, who died of phthisis.*

*The evidences of the growth of bone, though present, are slight (a). There is premature calcification around cell capsules (b). × 300 diamrs.*

Fig. 2.—*Deposit of nodular salts in the trabeculæ of a young bone affected with osteo-arthritis.*  
× 150 diamrs.



Fig. 1



Fig. 2





Similar lumps may be found in the granulation debris close to the articulation,—remnants probably of intra-cartilaginous or alveolar deposits. In proliferation nests that are, or have been, encircled by calcified material, nodules of various sizes may often be met with that have the optical and chemical characters of salts of lime. These have probably been set free by a liquefaction of the matrix in which they were embedded.

Thus we see that there are appearances not only of excess, but also of hurry and want of system in the process. Calcification takes place irrespectively alike of the preparedness of the cartilage cell groups, and of the ossifying process that is to succeed it. The salts are carried far up into the substance of the cartilage and thrown down around cell groups that have hardly begun to show linear proliferation. Masses of cells which are indistinguishable from marrow cells, and which, in health, ought to have been giving evidence of true bony growth at their periphery, continue to remain surrounded with calcareous formation. And, again, the development of bone may be seen taking place too soon with regard to calcification; or, what is the same thing, masses of salts which ought to have broken down and disappeared remain unchanged in the midst of the formed bone.

The salts are usually thrown down in their ordinary globular shape, larger and more irregularly formed than in health. Occasionally, however, appearances are met with which must be regarded rather as pathological accidents than as ordinary outcomes of the diseased state. Thus, spicular formations of the salts are seen, sometimes inside globes, like the crystals in a fat cell, sometimes in bunches like tyrosine. An exaggerated example of the latter form is shewn in Pl. XIV., fig. 2, where it crosses

## EXPLANATION OF PLATE XIV.

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Fig. 1.—*Pathological changes in articular cartilage at the edge of the outer condyle of a femur affected with osteo-arthritis. × 350 diamrs.*

- a. *Granular matrix.*
- b. *Cartilage capsules enlarged, and filled with highly refracting material in which are embedded irregularly formed cartilage cells and nuclei.*
- c. *A capsule and its contents which have undergone degeneration and proliferation.*
- d. *Bone, falling to pieces at e.*

Fig. 2.—*Peculiar brush-like formation of calcareous material stretching between articular cartilage and bone. From the trochlear surface of a humerus affected with osteo-arthritis. × 200 diamrs.*

- a. *Matrix with fibrillar calcification.*
- b. *A zone of homogeneous calcareous matter frequently found between cartilage and bone in young subjects.*
- c. *The brush-like formation.*
- d. *True bone and medullary cavities.*



Fig. 1



Fig. 2





over a clear band of apparently structureless calcareous material\* into the true bone. I doubt whether these formations are purely calcareous; but what their exact composition is I cannot say.

Excess in the amount of calcareous deposit is an early and common result of the diseased state. But it depends on the rapidity with which the changes advance as to how long the deposit will be permitted to remain. Calcification is less permanent than ossification; and, when the encroaching granulations invade the ossifying zone, the cartilaginous matrix, which bears the calcareous deposit, falls quickly to pieces, setting the salts afloat; while the formed bone may remain. As previously noted, this breaking up of calcified cartilage explains to some extent the diffused appearance of calcareous nodules in the more advanced states of disease.

In normal ossification of articular cartilage the individual cells and cell groups, after being surrounded with calcareous salts, suffer proliferative changes which leave them indistinguishable from marrow cells. In arthritic disease this proliferation is abnormal in position and excessive in amount. Thus, the cell groups may multiply and take on the form of medullary cells before they have been surrounded with calcareous deposits; and, before true bone has begun to be laid down in their periphery, several such proliferative nests may have become continuous. Again, in the healthy state, there is a sort of edge to edge meeting of calcified cartilage and bone. In the diseased condition the new bone appears around the pro-

\* A clear, highly refracting dense band of calcified material is pretty frequently seen at the junction of cartilage and bone in growing specimens. It is often broad enough to be seen with the naked eye. It is simply an exaggeration of the healthy process; a certain uniformity in density and regularity in breadth, giving to this portion of the calcified area an appearance of individuality which, on minute examination, it does not possess.

liferating nests at irregular and considerable distances from the old bone; and patches of true ossified material may remain for some time in the cartilage substance free from any connection with the bony mass underneath. In marked cases, there may be, over a breadth of several microscopic fields of three hundred magnifying power, an indiscriminate mingling of patches of bone and cartilage in the substance of the young proliferating cell growth.

By and bye, the granulations from the marrow advance in little buds and shoots into this area of newly formed bone and multiplying cartilage cells, and raise the cartilaginous mass from its already weakened bony attachment. But, while the mass of articular cartilage is thus severed from its osseous connection, the mixed layer, with its nodules of cartilage and young bone, remains embedded in the granulations. Thus, we nearly always find, even in cases of advanced disease, where the superimposed cartilage has entirely disappeared, that there remain with the nodules of bone in the granulation tissue, little pieces of cartilage in various stages of degeneration. The peeling of the cartilage is not therefore complete; pieces are left behind in the pathologically changed marrow.

Thus we arrive at the conclusions that the first effects of chronic osteo-myelitis on the bone-producing function of articular cartilage are to give it a temporary and unhealthy increase; and that the final results are a deterioration and ultimate abrogation of this function. But, under special conditions, it may happen that evidences of increase of osteogenetic function are absent from the beginning. This occurs when there has been made an early and efficient outlet for the proliferating marrow at a point in the articulation distantly situated from the piece under consideration; or where the inflammatory process



has been, from the beginning, slow and inactive, and degeneration has followed very closely in its train. There is, in fact, atrophy from the beginning. The cartilage has lost its healthy firmness and is easily stripped from the bone. There is a sluggish shrivelling and wasting of the cartilage cells, and a lowered activity of all the processes that indicate healthy bone formation. The deposit of lime salts is scanty; alveolation is incomplete; and true bony growth scarcely takes place at all. The condition is, in fact, an exaggerated example of what we find in the joints of very old people.

*Fibrillation of articular cartilage.*—Fibroid changes in articular cartilage, though much more frequent and important in the synovial form of disease, are yet sufficiently common in the disease under consideration to demand some notice. In chronic synovitis the fibroid transformation is brought about by a direct invasion of the granulation tissue into which the synovial membrane is changed. Synovial outgrowths eat their way into the articular cartilage from the periphery towards the centre of the surface, changing the cartilaginous into fibrous tissue as they proceed. No more need be said of the process at present, beyond this, that the fibres are derived from the cartilage cells, and that there is no fibrillation of the matrix. In the bony form of the disease the fibroid changes have a different origin, and the fibrillation is of the matrix as well as of the cartilage cells.

On the femoral cartilage of a knee joint in which acute suppuration had supervened on chronic osteomyelitis, I found several flakes of organised and fibrillated lymph. The flattened and normally sluggish cells near the articular surface could be seen passing by distinct

gradations into fibrillated embryonic tissue identical with the inflammatory exudation which overlay them. By invasion from the hyperplastic cells, as well perhaps as by liquefaction, the cartilaginous matrix gradually disappeared towards the young inflammatory growth. There can be no doubt that the final development of such a pathological change would be in a patch of complete fibrillation on the surface of the articular cartilage. I am doubtful, however, whether it is justifiable to postulate a similar origin for all the fibroid changes met with in this disease. No doubt attacks of more or less acute inflammation frequently supervene on the chronic course of the complaint; but it must be seldom, I think, that such attacks give birth to exudations of lymph. A more chronic process of fibrillation, claiming origin from the cartilage cells, is probably the rule.

The occurrence of fibroid transformation of cartilage cells is most common in very chronic examples of the disease. The change is usually met with as small white patches on the articular surface, lying, by preference, on the marginal portions. Most frequently the patches are quite superficial, but sometimes they extend to a considerable depth through the cartilage. I have never seen them in any way connected with the underlying marrow. Sometimes, especially on the patellar cartilage, large areas of the surface undergo the fibroid transformation. This is more liable to occur when the underlying disease is but slightly advanced. The cartilage then has enough vitality to express the irritation set up by contiguous disease in the comparatively healthy form of fibrillation.

The histological changes from cell to fibre are practically identical everywhere, and in no way materially differ from the healthy transformations, as from cartilage

to perichondrium. There is an elongation and tailing of the cells or nuclei within the capsules ; these, set free in the matrix, gradually become isolated ; they continue to elongate, become more and more crowded, and lie in planes more uniformly parallel to the free surface, an insensible gradation leaves them in the form of embryonic fibrous tissue, whence the transition is easy to formed fibrous tissue. In Plate XV. is depicted a section, taken from the patellar cartilage, which, if freed from surrounding portions, might easily pass for a healthy specimen of perichondrium.

In the above form of transformation the cells become fibres, the matrix disappears, and the result is pure fibrous tissue. But there is a fibrillation of the matrix which, in its fully developed condition, leaves a tissue homologous with fibro-cartilage. There is, of course, nothing extraordinary in this, when we regard the histology of embryonic articular cartilage ; and, in fact, of the matrix of all articular cartilage. We might consider fibrillation of the matrix either as an exaggeration of a normal condition or as a reversion to embryonic type. This form of change is found everywhere throughout the articular cartilage, more frequently, perhaps, at its periphery than elsewhere. The transformed areas are usually small in size. The fibrillation may be observed in all stages of advancement from an obscure striation to the development of a tissue which is almost indistinguishable from white fibro-cartilage.

What conditions determine the development of fibres in the matrix I cannot say. Though there can be no doubt that they are far more frequent in this disease than in health, yet I suspect that their occurrence is, to some extent, pre-determined by a tendency to the same

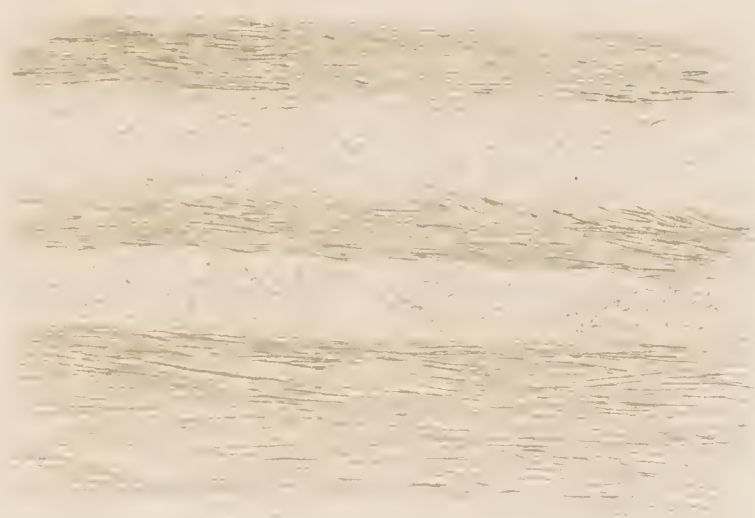


### EXPLANATION OF PLATE XV.

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*Fibrillation of articular cartilage, from the patella in a knee joint affected with advanced disease. The patellar bone was unaffected. The gradual change from cartilage cell to pure fibrous tissue is clearly seen. Near the top of the drawing is seen a vessel choked with blood corpuscles. × 300 diamrs.*







state before disease sets in. Be this as it may, fibrillation of the cartilaginous matrix, though of inconsiderable moment in the progress of the disease, must be described as a frequent but not essential concomitant.

*Breaking up and Absorption of Articular Cartilage.*—We have seen how the fungating marrow, in its efforts to find an outlet into the joint cavity, burrows under the articular cartilage, raises it in blisters, and ultimately eats its way through. We have noted how the cartilages are left in flaps or islands on the joint surface, and are gradually broken up in the debris of the destroyed joint. The changes which precede and accompany these net results have been traced and described. It still remains to follow up the concluding history of the cartilage,—the changes which are grouped between its severance from the bone and its disappearance.

The cartilage, though set free from its bony attachment, cannot be correctly described as necrosed. On the contrary, it is overfed. For, in place of the scant nutriment which, in health, soaked outwards from the bone, the fluid constituents of the blood now bathe it on all sides. The gross results are numerical hyperplasia of the cellular constituents, with disintegration, liquefaction, and ultimate disappearance of the matrix. The minute changes observed in the production of these results are varied, and may be described under the following heads :—

a. Simple excentric proliferation of cartilage cells, with concurrent absorption of matrix.

b. Linear proliferation of cartilage cells, with or without the formation of bloodvessels.

c. The formation of vascular loops which, in their advancing growth, convert areas of cartilage into granulation tissue.

## EXPLANATION OF PLATE XVI.

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### VASCULARIZATION AND DISINTEGRATION OF ARTICULAR CARTILAGE.

Fig. 1.—*Articular cartilage from the upper end of a tibia affected with osteo-arthritis. × 300 diamrs.*

*The cartilage cells undergo little change till they are actually absorbed in the young vascular tissue, and then they rapidly multiply. The transformation into granulation tissue is incomplete. The formation of a vessel wall out of the young cells is seen at v.*

Fig. 2.—*Articular cartilage from the head of a femur affected with osteo-arthritis. × 300 diamrs.*

*Proliferation within the capsules is seen in the neighbourhood of the degenerating area. The section was made a little above the apex of a tuft of advancing granulation material.*



Fig 1



Fig. 2





*Simple proliferation of cells and absorption of the contiguous matrix.*—The simplest form of degeneration of articular cartilage is where the cells inside the cartilage capsule undergo free multiplication, and the surrounding matrix breaks up into the proliferating centre. Consecutive subdivision of cells takes place with great rapidity, often giving, in a space not more than twice as large as the original capsular area, ten times as many cells correspondingly diminished in size. Most of the cells, even in their ultimate subdivision, retain a nucleus. Their contents are slightly granular, sometimes highly refrangible, and always contained in a well-defined cell wall. Cell groups in their vicinity become elongated towards the proliferating focus, and partake largely in the hyperplasia before they are incorporated in the general mass. Usually a number of cells are spindle-shaped; and, very frequently, well-formed fibres shoot inwards from the matrix and form a sort of reticulum. A surprisingly small amount of granular matter is present; the cartilaginous matrix seems to suffer almost perfect liquefaction and is completely lost sight of (Pl. XVI., fig. 2).

This form of degeneration is found in the thinnest pieces of cartilage, where there is most liberal supply of nutrient fluid. Thin margins of cartilage overlapping fungating granulations, or small isolated pieces that lie on their surface, give the best preparations. On the free surface of the cartilage a zone of this form of degeneration is of constant occurrence, and is frequently figured in books as “ulceration of cartilage.” But it is by no means confined to the cartilaginous surface; and the condition is only partially expressed by the term “ulceration.”

*Linear proliferation of cartilage cells, and advance of*

## EXPLANATION OF PLATE XVII.

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*Perpendicular section through a flap of articular cartilage partially detached from the condyle of femur affected with strumous osteo-myelitis.  $\times 350$  diamrs. Drawing one-third shortened.*

- a. *Granular debris and proliferating cartilage cells on the joint surface of the cartilage.*
- b. *Deep surface. Commingling of granulation tissue and proliferating cartilage cells.*
- c. *Linear multiplication of cartilage nuclei in direction transversely through the matrix.*
- d. *Multiplication of cells within capsules, some of the groups being in relation with the proliferating nuclear lines.*
- e. *Tailing of capsules towards a bloodvessel (not shewn) in the neighbourhood.*
- f. *Multiplying cell groups on the surface unconnected with the lines of nuclei.*







*granulation tissue in their track.*—In place of the general excentric proliferation just described, the cell groups may multiply only along definite lines. The direction of their growth is uniformly that of the nutrient currents, that is, transversely through the cartilage. Cell groups, still surrounded with capsules, are drawn out to many times their original length, and form anastomoses at acute angles with their neighbours. Occasionally these groups are drawn out to extraordinary lengths, as in Plate XVII, which is one-third shortened. And it is from marginal cell groups that these processes are sent through the whole thickness; the central groups take little or no share in their production. At the free edges, the cells, from which these proliferative tails arise, have already the structure of the granulation tissue into which they merge, or have made decided progress in that direction; in the central areas there is little change beyond a more or less marked tailing in various directions.

Plate XVII. represents as exactly as I could draw it a fairly typical example of this form of degeneration in its earliest stages. The long processes extending from side to side of the section are seen to be composed of minute flattened nuclei piled obliquely in single, double, or triple rows. In the lower portion of the section, these rows are derived from, or connected with marginal cell groups that have undergone free proliferation; in the upper portion no such connection is observed, though the cells have undergone the ordinary proliferation that is observed in the contiguity of over-abundant nutriment. Tailing of the capsules and multiplication of their cell contents is seen in the upper part of the drawing. This is an accident depending on the proximity of a vascular loop not represented.

The further history of this condition is as follows:—The chains of nuclei multiply rapidly, enlarge, and become indistinguishable from the surrounding granulation or medullary cells. Long channels full of rounded cells and granular remains of the matrix are thus formed through the cartilaginous mass. As cell multiplication goes on the matrix is encroached upon, the encapsuled neighbouring groups become involved, and burst into the channels. In this way considerable areas of cartilage may be destroyed without the appearance of bloodvessels. But usually, into the largest of the proliferating tracts the underlying marrow, with a bloodvessel or two, finds its way. In this case the solution and disintegration is more rapid and complete. The proximity of a medullary vascular offshoot can often be concluded from the evidences of increased vitality in the cell groups and nuclear chains, and from tailing of the cartilage capsules towards it instead of towards the free margins. Ultimately the cell growth and cartilaginous debris is either set free as pus in the joint, or remains as developed granulation tissue.

Elongation of cartilage capsules and proliferation of their cellular contents is frequently seen in conjunction with the above process. The termination of this change is also in anastomosing channels filled with cell growth. It differs from the preceding in the following points: the cartilage capsule is drawn out, and the cells multiply inside it; the channels are short and irregular in calibre; and the changes are seen in close contiguity to bloodvessels. In the preceding form the nuclear offshoots seemed to be sent out of the capsule; the channels, always long, sometimes attain to extraordinary dimensions, and are of nearly the same calibre throughout;



while it is characteristic of the process to take place at a distance from bloodvessels.

This form of disintegration is best seen in moderately thick portions of articular cartilage which have been set free, partially or completely, by burrowing granulations. I have found the best specimens where a flap of cartilage joined a fixed portion. Preparations in which the granulations have perforated the cartilage near the centre of the articulation, and where the flaps are attached round the periphery, are the most likely ones to afford good sections.

*The formation of vascular loops which convert areas of cartilage into granulation tissue.*—Unmistakeably distinct from the preceding varieties is this one produced by the aid of bloodvessels. In this instance the growth of the vessel is the close concomitant—we may say the direct cause—of the changes in the cartilage. The vessel walls are formed directly out of the cartilage cells, before those cells have taken on the form of perfect granulation tissue. There is thus a double connection between the vascular supply which causes the changes in the cartilage cells, and the cellular transformations which supply material to build the vessel with. In the previous instances the primary changes in the tissues took place at a distance from the vascular supply, and it was not till the changes were completed and the cells had become marrow cells that bloodvessels found their way into their midst.

The histological appearances are as follows (Plate XVI., fig. 1):—In a section well stained with carmine or logwood an island of slightly stained tissue, composed of rounded and spindle-shaped cells and short fibres, lying in a highly refractile matrix and traversed by a few capillary vessels, indicate the presence of the process. The

## EXPLANATION OF PLATE XVIII.

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### ABSORPTION OF CARTILAGE BY ADVANCING GRANULATION TISSUE.

Fig. 1.—*Isolation of a piece of articular cartilage by the meeting of tongue-shaped pieces of fungating marrow. Myelo-arthritic disease of the head of the tibia.  $\times 100$  diamrs.*

- a. *Healthy articular Cartilage. The cartilage cells next the marrow are in a state of proliferation.*
- b. *Underlying bone in various stages of atrophic degeneration.*
- c. *Fungating marrow abundantly supplied with blood-vessels.*
- d. *Nodule of isolated cartilage.*

Fig. 2.—*White fibro-cartilage from the side of the olecranon process of an ulna affected with myelo-arthritic disease. A vascularised tuft of granulation tissue has eaten its way into the fibro-cartilage in the neighbourhood of the joint cavity.  
 $\times 150$  diamrs.*

Fig 1



Fig. 2







cells are larger than white blood cells, have clear contents, a distinct nucleus and well-marked cell wall. In shape they exhibit transitional forms between the cartilage cells from which they are derived and the spindle-shaped cells which form the mass of the completely transformed tissue. The matrix in which they are imbedded is at first abundant, but gradually diminishes and ultimately disappears. Optically, the matrix is clear and glassy, standing out in prominent contrast to the granular and cloudy tissue which surrounds it. The fibres either rise abruptly from the matrix of the unchanged cartilage and run for some distance into the transformed tissue, or lie scattered indiscriminately through it. In the substance of the transformed area can be seen the building up of capillaries from the cells, which have still the clear refrangibility of the cartilage cells whence they are derived, and which are unmistakeably different from the granulation and exudation cells which give birth to capillaries elsewhere. At a little distance, still rounded; as they approach becoming spindle-shaped; and in the immediate proximity of the capillary, lying along its course, as long, fusiform, almost fibrous elements; there is no possibility of overlooking the steps of the process. The endothelial lining is an after addition. The completed transformation of the now vascular cartilage is simply, by easily understood steps, into ordinary vascular granulation tissue. (Pl. XVIII., figs. 1, 2.)

This variety of degeneration is best seen in thick, undetached pieces of young cartilage, and is particularly observable in cases of more than ordinary acuteness. Often, with the naked eye, tongue-shaped pieces can be seen creeping upwards into the healthy cartilage, having at their apical portions the minute structure above

described, and shewing towards their bases a more or less complete transformation into granulation tissue. Occasionally the buds anastomose.

In arthritic myelitis this invasion of the articular cartilage from below is the homologue, in scrofulous synovitis, of a similar invasion from above. In the synovitic disease it is probably the most common of all the changes. The final change, however, in the bony form of disease is granulation tissue and suppuration; in the synovial form it is nearly always fibrous tissue.

What influences are at work in determining the varieties of disintegration which articular cartilage undergoes would be a most interesting pathological study. The minute examination of nearly three dozen joints has not failed to afford many suggestions as to the pre-determining influences; these, however, I reserve till after a consideration of the other forms of chronic joint disease.

The profound disturbances of nutrition which occur in the presence of the disease under consideration give birth to changes in the articular cartilage which can hardly be classified under any head, and must be considered as pathological accidents. Among these one of the most striking is a tailing and contortion of the cartilage cells (Pl. XIV., fig. 1). The individuals in a mother group, after more or less extensive proliferation, assume peculiar, almost fantastic shapes in their beds of unusually abundant matrix. Some may be regularly rounded on one side and jagged and irregular on the other; some are notched all round; many send out curiously contorted processes, which sometimes subdivide, and suggest efforts at anastomosis with similar processes from neighbouring cells; altogether an endless

variety of form is observed. The nuclei are of all forms and sizes, mostly granular and non-nucleolated.

This change is probably allied to an appearance occasionally found in cartilaginous tumours, first described by Müller and afterwards by Paget.\* This form of cartilage cell, according to Paget, occurs normally in no vertebrate, but is similar to that found in the Cuttlefish. If I rightly interpret the drawings and descriptions in Paget's work, the cells he speaks of, though very similar to, are yet not identical with those in the cartilage of the Cuttlefish. In the pathological condition the tailed processes rarely or never reach beyond the enlarged mother capsule; in the Cuttlefish, however, their distribution is almost entirely in the matrix, and ramification does not take place till the processes have got outside the vaguely defined capsule.

A closer resemblance is to be made out in the following form, which, however, must be very rare, for I have met with it only once. Here the cartilage cells were represented by small, round, well-defined, slightly granular, non-nucleated bodies, distributed in thinly set groups through the matrix, and giving off a varying number of long, ramifying and occasionally anastomosing processes (Pl. XIX., fig. 1). The processes were evidently hollow, and contained particles which might claim descent from the original cell substance. The branching tubules were of almost uniform calibre till they tailed away unperceptibly into the matrix. At some parts they attained to a striking parallelism, and at one spot were almost as closely set as dentinal tubules (Pl. XIX., fig. 2).

The resemblance to the cartilage of the Cuttlefish is here striking and unmistakable. The similarity is car-

\* Lectures on Surg. Path., Vol. II., p. 178.



## EXPLANATION OF PLATE XIX.

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### PECULIAR PATHOLOGICAL CHANGES FOUND IN THE OUTER PORTIONS OF THE ARTICULAR CARTILAGE ON THE OUTER CONDYLE OF A FEMUR AFFECTED WITH OSTEO-ARTHRITIS.

Fig. 1.—*The cartilage cells send out long-tailed, branching, and occasionally anastomosing processes, which take on staining as deeply as the cells themselves. × 350 diamrs.*

Fig. 2.—*The same as Fig. 1, but that the tailed processes run nearly parallel courses. × 350 diamrs.*

Fig. 3.—*A section in the neighbourhood of Fig. 2, magnified 1000 diameters. Anastomosing channels, containing rounded and granular bodies, traverse the cloudy matrix in all directions. With the highest powers, no relation between the tubes and the cartilage cells could be made out.*



Fig. 1



Fig. 2



Fig. 3





ried into the ultimate elements of the tissues; in fact fig. 1, Pl. XIX., might be given as a correct delineation of the cartilage of the cephalopod.

From the situation in which this condition was found—at the margin of the articulation where processes from the connective tissue cells of the capsule merge into the cartilage cells proper—it would seem that it has some relation with the normal histology of the part. That the tubes are functionally nutrient canals there need be no dispute; but that they are in any way related to the questionable nutrient channels which are said by some histologists to pervade all cartilage I cannot believe. In the form of cell-tailing corresponding to that found in enchondromata we should probably see an indication of oncoming myxomatous transformation. This latter form is most likely to be looked upon as a pathological exaggeration of a normal condition.

*Note on Treatment.*—On the pathological views above enunciated it is easy enough to build up a plan of treatment. The chief obstacle in practice is the difficulty of forming a correct diagnosis in the early stages of the condition. Perhaps careful clinical observation will help to tide over this difficulty; and, thereafter, a considerable experience of the results would be necessary to give a firm footing to the treatment I advocate.

Of the condition called “tubercles in bone,” except as an integral part of general miliary tuberculosis, I know nothing. If it is a part of general tuberculosis then it is incurable, in the same sense that general tuberculosis is incurable; if nodules of caseation, analagous to patches of broncho-pneumonic degeneration in the lung, then they are an outcome of chronic inflammation of the marrow,

the condition I have been treating of. To the latter condition the name tubercle, in the more modern, and, in my belief, the more legitimate, sense, is not applicable. The caseation and encysting of an inflammatory patch in marrow is the best termination that can happen to it, except resolution. But this termination is so rare that it ought not to be waited for, if any satisfactory plan of treatment could be proposed. And when it does take place it will probably be unknown to us, for caseated masses are nearly always small and follow slight attacks of inflammation which give birth to few or no symptoms.

Following the lesions in chronological order we arrive at the following plans of treatment:—

In the beginning we find cellular hyperplasia, with the added circumstance that the swelling inside a non-expandible shell tends to strangulation. If we are to succeed, as in our treatment of scrofulous glands in the neck and elsewhere, by constitutional treatment alone, we must place the diseased bone glands under the same conditions. This means that, in the first place, they shall have room to expand. For this end,—to provide an outlet of escape for the hyperplastic elements,—an opening must be made in the compact shell. Then, having reduced to a minimum the danger of necrosis from compression, we might treat the case in the ordinary way with medicine and rest, and wait. The danger of laying open marrow cavities antiseptically is small, and is not to be counted against the danger of leaving the disease to its own course.

When the disease has made some progress towards the cartilage in one direction and along the shaft of the bone in the other it is still possible, without entering the joint or removing much of the cancellous bone, to get rid of most of the diseased tissues. A channel having been



drilled through the diseased centres with a small gouge or an elongated Volkmann's spoon, it is possible by injecting fluid with moderate force to wash out much of the hyperplastic tissue. Thereafter we might reasonably expect renewed vitality and healthy action.

In the more advanced conditions the cartilage and surrounding periosteum will have become affected. At first the changes in these tissues are conservative and help out the treatment. But the cancellated bone will have now begun to suffer from a decay which is probably irreparable, and nothing is left but to remove it along with the fungating marrow in which it lies. A Volkmann's spoon or a gouge carried through the opening in the compact bone would effect this.

In the final stages it is improbable that any treatment short of excision or amputation will suffice. But it ought to be borne in mind that, even after the cartilages have gone, there is still some hope of new bony tissue being developed from the encircling periosteum and of recovery taking place with fibrous ankylosis. This, of course, could not be expected to occur unless all the diseased structures had been removed. It unfortunately happens that, at this stage, the synovial membrane has become implicated, and it is probably in the experience of most that the pulpy synovial tissue is more difficult to get rid of even than carious bone.

Specially to be recommended is this form of treatment in disease of the tarsal and metatarsal bones. The possibility (which I have proved in my own practice) of filling the cavity left after removing the diseased tissues with healthy blood clot, which organises into fibrous tissue and bone, is more likely to be fulfilled in the smaller bones than in the larger ones. However, we

need not despair of filling such a large cavity as the condylar end of the femur, for as skill in the employment of antiseptics improves so does ability to carry out such delicate processes as the organising of blood clots.

Though my experience of the form of treatment advocated has been as yet small, it has been so favourable as to convince me that it possesses solid claims to recognition. The practice is a legitimate deduction from the pathology, and has, so far, answered well; how frequently, in the midst of the multiple conditions which surround every case of the disease, it is destined to be successful, remains to be seen.

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## THE MODE OF GROWTH OF SPICULAR OSTEOPHYTES.

BY MR. GREIG SMITH.

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SPICULAR or needle-shaped exostoses are never found as independent developments, but always in connection with other morbid growths. Consequently, an enquiry into their mode of production involves, at the outset, an examination of the processes wherewith they are associated. These may be summarised as follows :—

1. They are found in the substance of tumours, chiefly of the sarcomatous type, lying between periosteum and bone. Three such specimens are in the Bristol Infirmary Museum, one is in the Museum of the College of Surgeons of Dublin, and one in the Museum of the London Hospital. Four are connected with the bones of the skull ; in one instance, they sprang also from the ilium ; in one from the humerus.

The first of the Bristol Infirmary specimens is a macerated preparation of the whole skull, the calvarium of which is almost completely covered with a thick set crop of beautifully fine spicules of bone, varying in length from a few lines up to an inch. At some parts they are as closely packed as the bristles in a hair brush ; at other portions they are less thickly distributed. The calvarium on which they rest is rarefied and worm-eaten, here and



there, but is, on the whole, fairly healthy. The preparation was got from a child of thirteen, who died from the effects of an enormous tumour of sarcomatous character occupying the whole of the vertex of the skull.

The second of the Bristol Infirmary specimens shews the tumour and osteophytes *in situ*. It was taken from a man, aged 43, and consists of the parietal, and part of the frontal bones, with the tumour occupying both their aspects, and perforating the parietal bone near its centre. A moderately abundant crop of spicules grows from the unaffected bone into the tumour substance. The patient in this case had secondary deposits in the temporal bone of the opposite side, in the sphenoid bone, in the occipital, around the foramen magnum, and in the bodies of the upper cervical and lower dorsal vertebræ. The tumour structure was everywhere sarcomatous, round, and spindle-celled; the round-celled tissue largely predominating. In the secondary growths osteophytes were sparingly distributed.

The third of the Bristol Infirmary specimens is a macerated preparation of the humerus of an adult. Nothing could exceed the delicacy and beauty of the new growth. The upper three-fourths of the shaft of the bone is covered all round with a thick-set formation of fine spicules and diaphanous lamellæ. They stand out at right angles to the surface, varying in length from half an inch to over two inches. The humerus itself is very slightly affected. The osteophytes were embedded in a large sarcomatous tumour which completely surrounded the whole length of the bone.

The specimen in the London Hospital Museum is figured and described by Mr. F. M. Mackenzie in the fourth volume of the London Hospital Reports. The



patient was a boy three years old. The tumour was of semilunar shape, and sprang from both sides of the calvarium. The bony part of the tumour consisted of "vertical plates of thin bone attached at their thickest part to the outer table of the frontal bone, and gradually becoming thinner, ending at the circumference of the tumour in fibrous membranes. Between the plates was a quantity of soft, brain-like, and very vascular substance. The front boundary consisted of pericranium." The minute structure was that of a round-celled sarcoma. The lamellar structure apparent on section, is, as we shall see, a further development of the spicular.

The specimens in the Museum of the College of Surgeons of Dublin are mentioned by Mr. Hutchinson in the same volume of reports. "One of them is a part of one ilium, and the other a part of the skull. Both were removed from the same patient, a boy. On each bone there is a large patch from which bony fibres stand up erect like the hairs of a brush. \* \* \* The new growth appears to take place between the bone and its investing membrane (periosteum, pericranium, or dura mater, as the case may be)."

2. I have twice found diminutive specimens of spicular exostoses at the margins of sub-periosteal collections of pus; one on the cranium, the other on the shaft of the tibia. In both cases a sub-acute abscess had formed in connection with old-standing bony disease. Though the spicules were small and not very closely set, there could be no doubt as to their origin and character.

3. With some hesitation I mention their occurrence in connection with true cancer of bone. An old preparation in the Bristol Infirmary Museum shews imperfectly formed spicules around the orbit, which is said to have

been occupied with a cancerous tumour. This preparation I should have passed over, had I not met with another, where, in a patient that died with typical cancer of the liver, there was a cancerous tumour in one of the ribs, in which were embedded fairly good specimens of spicular osteophytes. Of this case I have no notes, and I quote it from memory.

No doubt other museums contain similar specimens; and it is very probable, as we shall see, that many forms of exostosis which have a more or less close resemblance to the spicular form, were, in their original development, entirely of this variety. In an investigation as to their mode of origin and growth, I have thought it wise to confine myself to specimens which were as nearly as possible typical.

Now, in all these specimens I have noted, we have this character in common, that the periosteum was stripped bodily from the bone. There is certainly some amount of uniformity in the character of the tumours with which this form of osteophyte is associated; but the uniformity lies more in the position they happen to occupy, viz., between periosteum and bone, than in the histological elements of which they are composed. Starting on this hypothesis, that they are somehow connected with the stripping of periosteum from bone, the possibility of shreds of periosteal fibre, or periosteal vessels on the stretch, serving as parent tissue for bony growth, immediately suggests itself. Indeed, at the periphery of the new growth, by carefully peeling off the periosteal membrane, fine threads of its substance may be seen running down to the tips of the minute spicules which spring from the compact shell. Billroth seems to have found this; and suggests, rather than describes, their

connection with periosteal vessels. It remains to be seen how far these shreds, vascular or simply fibrous, are concerned in the origin of the bony spicules in question.

These shreds are directly continuous with, and structurally indistinguishable from, the fibres of the overlying periosteum. Adherent to their surface and infiltrating their substance are numbers of rounded and fusiform cells, similar to those found in the so-called "cambium layer" of the periosteum. Frequently, but not always, a bloodvessel runs up the centre of the thread, being lost in the periosteum on one side and penetrating the tip of the spicule on the other. It is evident, in fact, that the spicule of bone follows this thread of fibrous or fibro-vascular tissue, but it is difficult to make out their precise structural and developmental relations. The bone and fibre are both too thick to be studied with a high power, and too delicate to be cut longitudinally with the razor. I had therefore to be content with sections through tumour, bone and periosteum, which give only fragmentary views in individual specimens, but, in conjunction, give a sufficiently adequate comprehension of the processes at work.

We know well enough that inflammatory hypertrophy of bone takes place, not by virtue of the inherent capabilities of the osseous tissue proper, but through the medium of some parent tissue. In this case it would make matters simple enough to say that the periosteal shred with its groups of cells was the parent tissue, and that the bony growth followed it. But this simple statement will not suffice. In the first place it takes for granted the process that wants to be explained; and, secondly, exception might be taken to it on the ground that the ossified spicule, hollow and arranged in concen-



tric lamellæ, bears no resemblance to bone developed purely in fibre.

A large number of minute vessels run between periosteum and bone. The tunica adventitia of a periosteal artery is a direct continuation of the periosteum as far as its entrance into the bone; but, in the medullary cavities, its fibres are spread out and become fused with the fine areolar framework which supports the fatty tissue and proper round cells of the marrow. In the borderland between periosteum and bone it is surrounded by, but not infiltrated with, the cells of the cambium layer. Inside the bony substance the fibres of the vascular walls, now much more attenuated, are freely infiltrated with the cells which serve the purpose of osteoblasts in the medullary cavities.

Now, let us suppose that an advancing tumour is stripping the periosteum from the bone. In the retiring angle between the membrane and the bone will be found proliferating "cambium" cells, mingled with numbers of exudation and connective tissue corpuscles—outcomes of the inflammatory process. Traversing this cellular aggregation will be found vessels and shreds of periosteal tissue on the stretch between the bone and periosteum. The vessels will be partly pulled out of the medullary cavities, partly dragged from the periosteum. As yet the tumour proper will not have invaded this part.

Around the periphery of the tumour these are the conditions which immediately precede the spicular bony growth. The first deposit of new bone takes place as an elevated ring on the compact bone surrounding the periosteal vessel, and is, in the first instance, a medullary development. Further on, where the fibres of the vessel wall are periosteal in character, the new growth is of the



periosteal type. That is to say, the base of the spicule resting on the bone is composed of concentric lamellæ arranged round the central vessel and medulla; the apex is built up of a solid structure not arranged in lamellæ, and with that absence of regularity in the arrangement of bone corpuscles which marks the primary growth from membrane. A hollow cone is thus erected around the vascular opening, and increases in size after the manner of normal bony growth. The periosteal thread leads the way and the marrow follows. Periosteal bone, with its irregular arrangement of bone corpuscles and want of homogeneity in matrix, is laid down in the substance of the fibre, in which, as we have seen, all the materials necessary for its formation already exist. In the tract of this deposit the marrow follows, absorbing, alveolating and re-depositing in lamellæ. Thus the spicule is built up. A more or less solid apex of irregular and perhaps areolar bone, resting on a hollow cone built of concentric lamellæ around the opening in the compact shell; the structure at first traversed by a bloodvessel, which usually, from stretching or pressure, becomes occluded; the hollow portion filled with ordinary medullary tissue, nourished by vascular outgrowths from the underlying bony marrow, is a summary description of the spicular osteophyte.

As growth advances new Haversian systems are developed inside the cavity of the spicule, and more or less completely formed secondary ossicles are laid down on its inner walls. These go on developing after the manner of medullary bone by absorption and re-deposition, and soon the interior of the cone is filled with lamellar septa, on the walls of which a third formation of small hollow tubes or ossicles are found. And so the growth goes on,

each spicule an exact epitome of the ordinary development of a long bone.

Through an excess of the medullary over the periosteal growth the exact spicular shape in the end disappears. As alveolation takes place inside the cones, and neighbouring spicules increase in size, their walls come into close contact, and ultimately their cavities open into each other. By a continuation of the absorption of contiguous walls and coalescence of cavities the bony lamellæ and medullary cavities may reach considerable dimensions. In the meantime the periosteal thread has either disappeared or, in the presence of the active growth that has sprung up in its tract, has lost its functional importance. Solid apices on the spicules are no more seen, and the medullary cavities open freely into the encroaching tumour substance. At this stage the exostosis loses its title to the term "spicular," and ought rather to be called "lamellar." It is seldom that the lamellar development reaches the advanced stage of the case quoted above, but the first steps of the process may be seen in most preparations.

To sum up—

1. Spicular osteophytes originate in the retiring angle between periosteum and bone, when periosteum is stripped from bone by a growing tumour.

2. They are partly periosteal, partly medullary in origin. The periosteal tissue is usually the adventitia of a stretched vessel; the medullary is derived from a vascular opening in the compact bone.

3. The apex of the spicule, developed in fibre, is solid or areolar; the base, developed from medulla by alveolation and re-deposition, is hollow, and composed of concentric lamellæ.

4. In the end the medullary predominates over the periosteal growth. The character of the exostosis is then rather lamellar than spicular.

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## EXPLANATION OF PLATE XX.

ILLUSTRATIVE OF THE GROWTH OF SPICULAR  
OSTEOPHYTES.

*The section (A) is supposed to have been made at the margin of a tumour lying between periosteum and bone. The tumour structure is not represented.*

*Fig. B is a diagrammatic illustration of the more advanced growth of the spicules.*

- A. a. *Healthy periosteum.*  
b. *Cellular layers lying between periosteum and bone.*  
c. *Healthy compact bone.*  
d. *Periosteal vessel entering bony foramen, and surrounded with an adventitia derived from periosteal fibre.*  
e. *Periosteal vessel put on the stretch. Between d and e, the advancing tumour is supposed to have stripped periosteum from bone.*  
f, g, h. *Periosteal vessels getting more stretched and ultimately occluded.*  
k. *Pink marrow.*  
l. *Haversian canal.*  
m, n, o. *Shewing the progressive development of bone from fibre and marrow.*
- B.—*Transverse section of four hollow spicules, which by continuous growth and absorption of contiguous walls would assume some such form as the triangular outline indicated below.*







# THE PROGRESS OF PUBLIC HEALTH IN OUR OWN TIMES.

BY DR. BEDDOE.

Presidential Address before the Bath and Bristol Branch of the British  
Medical Association, 1879.

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IT is usually the most difficult duty of the President of a scientific society to select a subject for his opening address; and I have felt the difficulty on the present occasion, notwithstanding, or perhaps rather in consequence of, the excellent examples set me by past Presidents. He would be a bolder man than I who would undertake to give his brethren a survey or epitome of a year's progress in our extensive, manifold, and fluctuating art. Papers on special subjects, on which the readers may think themselves able to communicate information to others, are best reserved for discussion at ordinary meetings. More suitable, I think, are such general subjects as afford some scope for originality in treatment or exposition, or such as relate to the history or politics of the profession or society.

I have accordingly chosen for my theme "The Progress of Public Health in our own Times," and propose to treat it from the statist's rather than from the sanitarian's point of view. Materials for the purpose are copious if not altogether satisfactory. Mr. Graham and

our own Dr. Farr have given us for forty years past the best and most complete system of mortality statistics that exists in any country; and if their publications leave anything to be desired, it is certainly not their fault. The Scottish mortality statistics are also excellent: the Irish, owing to the weakness of the law, are scarcely available. Some of our colonies publish elaborate and trustworthy statistics; those of Victoria, in Australia, as edited by Mr. Archer and Mr. Hayter, would do honour to any country. In the United States this department is strangely neglected; but several European states vie with us in accuracy, if not in copiousness and variety of facts.

It is therefore possible to adopt the lateral as well as the historical method in investigating the progress of public health in England, seeing that we may find in some countries, notably in the east and south of Europe, social conditions and rates of mortality resembling, and throwing light upon, those which obtained among ourselves two centuries ago; while in others we may find conditions to be envied and to be emulated.

Since 1838, when our registration system was founded, considerably more than a generation has passed by, and great material and economic changes have taken place. The great development of our railway system, the general adoption of gas-lighting, and the triumph of free trade, were all subsequent to 1838. Consequent on or connected with these have been a decrease of arable cultivation, and therewith of the number of agricultural labourers, the most healthy class of men in the country, a great increase of factory and associated labour, of mining and metal-working, and of nocturnal labour. On the whole the lower classes have been more highly paid,



they have consumed more animal food, and apparently also more alcohol, tea and tobacco ; and milk, that great blessing to children and invalids, has become more easily procurable in towns but less so in the country. An immense variety of small luxuries and comforts, easances for the old and feeble, have come into vogue. Dry-nursing has become a comparatively easy matter, since the invention and improvement of the feeding-bottle ; but, per contra, the natural mother appears to be more and more failing in ability to discharge this part of her duty. But the greatest and most important, perhaps, of all these changes are the rapid increase, partly intrinsic, partly by immigration, of the population of our towns, and the diminution, not merely comparative but actual, of that of the open country. As a rule, the larger the town, the more rapidly it increases up to a certain point, and thus not only is the countryman becoming a townsman, but the townsman is becoming a citizen—the aggregation which is the essential quality of urban life is becoming not only more general but more intense. It is not easy to give very clear expression to its amount ; but in Scotland, where it is progressing *pari passu*, the eight principal towns, which twenty-four years ago contained about twenty-eight per cent. of the whole population, now embrace thirty-five per cent. In England and Wales, again, during the forty-two years from 1837 until now, about ten millions have been added to the population, which had been only fifteen millions ; and we may safely say that the whole of this increase of ten millions has been added to the towns. In fact, the English people, who during the middle age and even much later, were distinguished from most continental peoples as dwellers in the open country or in small hamlets rather than in

towns, and possibly owed some of their undoubted physical superiority to that fact, are now rapidly becoming a nation of citizens, and indeed are so already in greater proportion than any other European people. France, however, is following pretty quickly in our track ; and in almost every State in Europe, even in sparsely populated Norway, the capital and a few centres of commerce and industry are growing rapidly.

The citizen endeavours to escape from the increasing pollution of air and water by colonizing the environs, but though he to some extent obtains his object, the suburbs thus created, being constructed without system or control, tend after a time to degenerate in healthiness. The great countervailing influences, however, which war against the evils caused by the aggregation of mankind, are the sanitary and philanthropic movements, both of which, we may fairly boast, have had their origins, and have attained their greatest proportions, in our own country. Into their various developments we have not now, of course, time to enter ; but it may be well to mention the general extension of the water-closet system, the appointment of sanitary boards and medical officers, the war begun against zymotic diseases by isolation and disinfection, and against dietic diseases by the adulteration acts and similar legal measures. I may here perhaps mention one of the latest advances made in this war, by a detachment under the command of Capt. Sir Robert Christison. A voluntary association has been founded in Edinburgh, at the suggestion of Professor Fleeming-Jenkin, for the inspection of private houses by a skilful engineer, rectification under his eye (so far as that is possible, but, alas ! scamping workmen may defeat the quickest eye), and subsequent annual or semestrial re-examination. Of 413

houses hitherto inspected, only 33 have been found blameless, or about 8 per cent. Changes in the method and plan of building have not all been for the better: the speculating builder is certainly more of a curse than a blessing to the country; official inspection is generally a mere farce; and in cottages warmth is often sacrificed to considerations of decency and convenience.

The great extension of thorough drainage in the open country, though carried out with purely economical views, has affected human health and life by drying the soil, as well as by aggravating floods.

Of social changes affecting health, mental or bodily, the principal are the recent educational movement, including the extension of competitive examination, the increasing popularity of athletics, and the increase of early marriages. The average age of marriage, indeed, has remained pretty constant for the forty years under review; and if the customary or usual agreed with the average time, it would be well for us, for in the case of spinters it is 24 years, or exactly what Dr. Matthews Duncan has fixed on as the best from a hygienic point of view. The fact is that too early and too late marriages have both increased, and to such an extent that they statistically balance each other. In the case of men, or rather boys, the proportion of minors marrying has risen from 4 to 8 per cent., and in that of women from 13 to 22. So much for the agencies which may have affected the public health of England, for good or for evil, during the past forty years. Let us now investigate our actual position, as indicated by the rate of mortality.

The last three years have been extremely favourable in this respect. They have been years of copious rain, of scanty harvests, of bad trade, and, whether in spite or in



consequence of all these, of low mortality in both England and Scotland. It is a sign of England's great wealth and sound economical laws, but also perhaps of her moral degradation, that times of poverty and distress, which in some countries raise the death-rate, do sometimes in England, as during the Lancashire cotton famine, have the contrary effect. It will be fairer to stop at the close of the year 1876, and more convenient, too, for several reasons.

In 1876 the death-rate of England for both sexes was 21 per 1,000; for males, 22·5; and for females, 19·7. That of Scotland was also 21. But if we take the four years 1873-76, England will yield us 21·8 and Scotland 22·5. The rate for Ireland would no doubt be lower than for Great Britain.

Forty years ago it was believed, perhaps wrongly, that the expectation of life was higher in England than in any other country. It certainly is not so now. The following is the order of the death-rates, for the same four years, in several countries of Europe:—

Norway comes first, then

Sweden ...	...	...	...	...	...	...	19·3
Denmark ...	...	...	...	...	...	...	19·8
Belgium...	...	...	...	...	...	...	21·6
England ...	...	...	...	...	...	...	21·8
Scotland ...	...	...	...	...	...	...	22·5
France ...	...	...	...	...	...	...	22·6
Netherlands ...	...	...	...	...	...	...	23·8
Prussia ...	...	...	...	...	...	...	25·7
German Empire ...	...	...	...	...	...	...	27·2

The lower position of Germany than of Prussia is mainly due, probably, to the inclusion of Bavaria.



Austria, Italy and Spain, about	...	30
Russia, more than	... ..	30

In our Australasian colonies, where the population is a mixture of British races in nearly the same proportions as obtain in these islands, subjected to climates for the most part warmer and drier, the average death-rate in 1875 was 18·6. This is a very low rate, even for a rapidly increasing population with an unfairly small proportion of old people. In New Zealand, where the proportion of immigrants and of births attains its maximum and the climate most nearly approaches our own, the rate was only 15·9; in Tasmania, with a stationary population and fewer births than in England, it was 20; in Queensland, under the tropics, 23·8. In the healthier year 1874 all these rates had been considerably lower. Clearly, therefore, the race is capable of better things under better conditions.

A fairer comparison may be made by ascertaining the population and death-rates at different ages and summarising the result. This has been done by Bertillon,\* who finds that on this plan France, where children are fewer, takes a better position than England, the order of thirteen European States being as follows:—

- |                 |                  |
|-----------------|------------------|
| 1. Norway.      | 8. Holland.      |
| 2. Belgium.     | 9. } Austria and |
| 3. Switzerland. | 10. } Spain.     |
| 4. } Sweden and | 11. Bavaria.     |
| 5. } France.    | 12. Italy.       |
| 6. England.     | 13. Russia.      |
| 7. Prussia.     |                  |

Except in infancy, and from 15 to 30 years, England

\* *Demographie figuree de la France.*

comes out behind France; and our position is most unfavourable, as might perhaps have been anticipated, in middle life, the period of hard work, from 30 to 60 years.

The mortality of our large towns, as a rule, compares creditably with that of the great cities of the Continent, especially those of Sweden, Russia, Germany and the South of Europe, where the rate is generally over 30 per 1,000, and in some instances, as at Prague, Budapest and Odessa, even over 40. Yet even here our superiority is less distinct than it at first appears. Thus Manchester is inferior by every test to Ghent, its Belgian counterpart; and Manchester, Liverpool and Glasgow are all inferior in the expectation of life at most ages, even to so notoriously unhealthy a city as Berlin;\* while in the proportion of their inhabitants who die beyond their 75th year, *i.e.*, who reach the period of life when most deaths take place in healthy districts, they fall below even Prague, and scarcely surpass Moscow itself.

Here we may note a curious and important fact, and one of favourable augury. It is this, that though the collection of people into cities increases the mortality, and though that increased mortality is, roughly speaking, as Dr. Farr has demonstrated, in the ratio of the density of population, it by no means follows that the larger the town the worse will be the death-rate. Thus London is notoriously healthier, so far as mortality goes, than any town of the second magnitude in England, with the doubtful exception of Bristol. It is perhaps hardly necessary to quote figures, but I may remind you that the death-rate of London, for the ten years ending 1876, did not exceed 22 per 1,000, while that of all the other urban districts, taken together, was about 25. Nor does this

\* *Statistique Internationale des Grandes Villes*, Vol. I. Budapest, 1876.

OF 10,000 DEATHS, THE NUMBER OCCURRING AT SEVERAL AGES.

	Under 1.	Total under 5.	5+	10+	15+	20+	25+	35+	45+	55+	65+	75+	85+
Manchester, with Salford and Charl- ton, in 1872, males }	2716	4840	453	184	252	299	641	792	814	812	624	275	43
Ditto, females .....	2663	4851	462	181	264	321	699	786	837	855	683	321	39
Prague--Males, 1865 } to 1874 .....	2870	3831	197	128	347	525	947	953	1007	865	791	342	74
Ditto, females .....	2550	3534	201	124	358	663	1075	823	848	845	884	516	131
Moscow--Both sexes, } 1865 to 1874 ... }	2924	4043	242	195	333	475	1021	1072	897	768	627	344	



superiority depend, unless in part, on any peculiarity connected with the situation of London, or its character as a capital city. For in Scotland we find that the eight towns of largest population, technically called "the principal towns of Scotland," namely, Edinburgh, Glasgow, Dundee, Aberdeen, Greenock, Paisley, Leith and Perth, have of late years yielded a death-rate considerably lower than that of towns of from 10,000 to 25,000 inhabitants. Last year the difference in favour of the "principal" towns amounted to nearly a half per cent., their mortality being 23·7 per 1,000, against 28·0 in the "large" towns, as the Registrar-General for Scotland calls the other class just mentioned.

This somewhat paradoxical phenomenon reappears in France, though less markedly. The mortality of Paris for the last three years is stated at 25·4, and the general urban mortality of France at 26·1. I cannot check this statement; but I have little doubt of its correctness; for a similar relation subsisted during the fifteen years prior to 1850. The death-rate of Paris was then 26·7 per 1,000, that of the capitals of arrondissements, many of them quite small places, was on the average 26; but of 48 cities all exceeding 20,000 in population, 33 stood worse than Paris in this respect, four were even with it, and only eleven stood better than Paris. One of the eleven, I may add, was the very English town of Boulogne.\*

\* In the following cities the rate of mortality exceeded that of Paris. The order is merely geographical :—

Lille, Valenciennes, St. Omer, Arras, Amiens, Reims, Troyes, Strassburg, Colmar, Versailles, Rouen, Cherbourg, Angers, Rennes, Brest, L'orient, Tours, Orleans, Bourges, Dijon, Lyons, St. Etienne, Clermont, Limoges, Rochefort, Bordeaux, Grenoble, Aix, Toulon, Marseilles, Nîmes, Montpellier, Persignan—33.

In the following the rate fell below that of Paris :—

Cambray, Douay, Boulogne, Metz, Nancy, Caen, Le Mans, Nantes, Poitiers, Montauban and Carcassonne—11.



Even in Sweden, though Stockholm is an extremely unhealthy city (as unhealthy as it is pretty, just what Salisbury used to be among us), though Stockholm, I say, has a mortality considerably over 30, Gothenborg, Carlskrona and Jonköping, the towns next in size, are considerably worse.

The causes of this anomaly are not, I think, far to seek; doubtless they are complex, but the leading one is the centripetal attraction of capital, energy, intellect and medical skill towards the largest towns, whereby it comes to pass that these towns are the first to adopt sanitary improvements of every kind.

The birth-rate as well as the death-rate is usually lower in great cities, and this latter fact has some degree of influence upon the former one. I do not know that this is a subject of congratulation to us in Britain. Vitality, so to speak, burns itself out in cities, but so do intellect and beauty too. If we could rely on these being constantly redeveloped, in association with health, energy and longevity, in the rural districts, it might be well; but there is reason to think that the elevation of a race in most of these qualities goes on but slowly, and moreover it is not in the rural districts that births are most numerous and the springs of population well up most actively. Marriages grow fewer in the open country, and the ratio of births is generally low. The Scotch statistics, from their method of arrangement, are the clearest on these points. There the excess of births over deaths, the natural increase of population, rises gradually from the most purely rural and sparsely peopled districts up to that second class of towns which yields the highest mortality.

And in the following it was about equal:—Dunkirk, Havre, Bescançon and Toulouse—4.

The excess is as follows, per 10,000 living :—

	Insular Rural.	Edinburgh.	Other Principal Towns.	Mainland Rural.	Small Towns.	Large Towns.
1876 ...	103	113	125	145	155	160
1878 ...	105	123	139	148	162	183

The most prolific of these “large towns” are for the most part those most exclusively concerned with the coal and iron industry.

If we examine the English returns we shall find a similar state of things: the excess of births over deaths in the old county towns and cathedral cities, or residential towns, is comparatively small; but in the mining and metal-working towns and villages it is enormous, often exceeding 20 per 1,000 on the population. In Middlesboro’, in 1876, it was 26 on the estimated number of inhabitants. In Bedminster it usually reaches or approaches 20, and in some of the mining towns and villages of Glamorgan is still higher.\*

Doubtless the immense number of births is due in great measure to the crowds of young marriageable people who flock into these places from those where labour is less in demand. But the fact remains that these unlovely regions, whence “sweetness and light” are so completely banished, are the “*officinæ gentis*,” the breeding-grounds of the coming generation of Englishmen. Natural selection will bring through the perils and accidents of childhood the hardiest and coarsest-fibred of

\* In 1872 Dudley and Wigan, both places of unenviable reputation, had the highest birth-rates (46·9 and 46·1 respectively). The excess however of births over deaths was greatest in Guisborough (24·6), in Stockton (22·4), in West Bromwich (21·9), and in Barnsley (21·4). All these are coal-and-iron districts.

the brood; but we cannot expect that they will in all respects be an improvement on their peasant ancestors.

The subject of seasonal variations of mortality, which has a pretty direct bearing on the one I have now in hand, was much studied by Hippocrates, and little was added to his observations on the subject until near our own times. Of late Boudin,\* Farr, Oesterlen,† and quite recently Mitchell and Buchan,‡ have considerably added to our information upon it.

Our Registrar-General does not, however, enlighten us on it so much as he might. He prefers the quarter to the month in his division of seasons. It is not, therefore, possible to construct for England such curves of seasonal mortality from all or from special diseases as Mitchell has done for London and New York. But we know that, taking England *en masse*, the winter quarter is the deadliest, the summer the least deadly, and always so except in choleraic years; that the spring and autumn have for these forty years occupied an intermediate position, but that whereas spring used commonly to stand worse than autumn, of late their respective positions have as often as not been reversed. This seems to be one of the changes consequent on the rapid growth of towns, for in the rural districts, on the average, and in two out of three years, the old order (winter, spring, autumn, summer) is still observed, while in London and other large towns winter, summer, autumn, spring is the usual one.

In an average of fifteen years, ending with 1876, the proportions were as follows:—

	Quarter ending March.	June.	Sept.	Dec.	Year.
130 large town districts ...	25·5 ...	22·1 ...	22·9 ...	22·7...	<b>23·3</b>
Country and small towns	21·4 ...	18·9 ...	16·9 ...	18·1...	<b>18·8</b>

\* *Traité de Géographie et de Statistique Médicales.* † *Medicinischen Statistik.*

‡ *Journal of the Scottish Meteorological Society*, Nos. 43-46 and 55.



Mitchell and Buchan's mortality curve for London, based on the experience of 30 years, starts from a maximum early in January, dips a little in February and rises through March to a point very nearly as high, then descends rapidly through April and May to a minimum of deaths in the middle of June, then rises sharply to another maximum in July, not much lower than those of January and March, then declines slightly through August and more decidedly through September to a secondary minimum in October, and finally rises rapidly through November to attain about the maximum in December.

The secondary maximum in July is due principally to the prevalence of diarrhœal disease, of which the initial cause is excessive heat, the mortality invariably rising when the temperature keeps steadily above 60° F. (15·5 C). The comparative absence in the open country of the conditions through which the heat acts, the filth, the foul drains, the putrescent food, &c., renders the heat there almost innocuous, and nullifies the secondary or summer maximum. In Scotland the absence or mildness of the summer heat has the same effect. Even in the eight principal Scotch towns there is scarcely a trace of a summer rise except in unusually warm seasons, the most that can be observed in ordinary years being a little delay in the downward progress of the mortality curve towards its minimum, which is usually in September. The Scottish townsfolk die of cold and wet, underfeeding and overdrinking.

On the other hand, our own kindred when they migrate to colonies exposed to greater sun-heat experience their maximum of mortality in summer. In New York, where both winter and summer are severe or extreme, the maximum is in July; but this is entirely due



to the deaths of children under two years of age, which are so numerous as to conceal the maximum for all other ages, which is in winter and spring. In Australia, in all parts of which the winter is mild, the maximum is in summer, and the death-rate continues pretty high until far on in the autumn.

Not that the seasonal curve of mortality is determined simply by the compass and the thermometer. Prevalent modes of living affect it much; and the element of race is also important, race being, for this purpose, taken to mean not necessarily community of blood, but a likeness of constitution engendered and maintained by long periods of common habits and conditions. I suspect that the British constitution is more susceptible to what may be called zymosis and filth-poisoning than that of some other races. It certainly bears heat badly.

The seasonal or mensual maximum is very varied in the countries and cities of Europe, and by no means conforms to what one might expect. On the whole, spring seems to be the worst season in the greatest number, perhaps owing to the keen east winds. Scandinavia in general, France, Bavaria and other parts of Germany, have the spring maximum; but it seems to have shifted from spring to winter in Paris in the course of this century.

According to the well-known aphorism of Celsus, autumn was in his time and country the most dangerous season, and after it followed in order summer, winter and spring. That is to say, according to Dr. Farr, that malaria was more deadly than severe weather. This may still be true in some parts of Italy, but not, apparently, in the towns. Palermo, indeed, follows Celsus's rule; but Venice has a spring maximum, being much

exposed to the east. Rome and Turin agree nearly with London; and Naples, strange to say, has simply a winter maximum. The proportion of deaths in sunny Naples from pulmonary diseases is far greater than in London.

Yet more instructive is the fact that Iceland has its maximum in summer, the length of the summer day probably making up for the moderate heat, and co-operating with the dirty habits of life brought about by the want of fuel. In the cities of Russia, too, and several of those in central Europe, there is a high summer maximum, and the winter mortality is below that of spring. Here the short hot summer tells heavily on filthy and unsanitary conditions, and on constitutions hardened against cold.

Now we know that a few generations back the citizens of London, then the only great city in England, lived habitually in a state of noisome filth, which probably even Russia cannot now parallel. Accordingly the death-rate of London was fearfully high. In the seventeenth century, even in years when plague was absent, it is said to have been about seven per cent., and the seasons conformed pretty nearly to Celsus's rule, autumn and summer being more deadly than spring and winter, though even winter was twice as fatal as it is now.

(1606-10. Annual Morty., J, F, M, 1·4; A, M, J, 1·5;  
J, A, S, 2·7; O, N, D, 2·0; total, 7·0 per cent.

*Farr, second annual report.*)

Vulgar errors, we know, are often the discarded opinions of men of science. And sometimes they *have been* true, though they have ceased to be so. May not the still prevalent error that "a green yule makes a fat

kirkyard," have come down to us from a time when the unsanitary habits of our forefathers made warm relaxing weather in winter dangerous to them?

Improvement in the health of London has probably been continuous since the date of the great fire and the disappearance of the plague. At the beginning of this century the death-rate was 29 per 1,000; which is about equal to that of Manchester and Liverpool and others of our worst towns at present, and would be highly respectable in Italy, or even in Germany or Sweden. But the mortality of the open country and small towns of England (to judge from the Carlisle table), was already very moderate. The population was fairly housed and substantially fed, and the struggle for life and wealth not intense. In most European countries, meanwhile, poverty and scarcity pressed more or less, or from time to time, on the peasantry, and there was more room for improvement. In Sweden the expectation of life advanced five years in the last half of the eighteenth century; and during the last twenty years an improvement in the death-rate of from 1 to 2 per 1,000 has taken place in France, Belgium, Sweden, Prussia and the Netherlands; a considerable one, too, in Austria; but, probably, none in Hungary, Italy, Spain and Russia, which remain at the bottom of the scale.

Whether the public health in Great Britain, as measured by the death-rate, has improved at all during the past forty years, may admit of some doubt. That it has somewhat deteriorated in the northern part of the island scarcely admits of any; though the last three years in Scotland have shewn a tendency to amendment, specially marked in the great towns.

In England it would not be difficult, by mere manipu-



lation and classification of the years and the figures, to exhibit either an improvement or the reverse. The simplest, and probably truest, way of looking at the subject is to say that for forty years alternate rises and falls have taken place in the death-rate, which has oscillated between 20·5 and 25·1, generally in periods of elevation or depression lasting from two to five years, and roughly corresponding to cycles in the weather; and that we are now, it is to be feared, coming to the end of a favourable lustrum. Four or five years ago one would have had little hesitation in saying that the mortality of males had increased, while that of females was nearly stationary, or very slightly better. Just now one might fairly enough say that that of females had decidedly decreased, and that of males had been pretty steady. That the condition of women has improved relatively to that of men is perfectly clear.

The proportion of male deaths to 100 female deaths, out of equal numbers living, was forty years ago 108. It gradually declined to 105 in 1849, and has since then gradually risen to 113 or 114; and as the ratio of male to female births has meanwhile lessened from 105 to 104, it is clear that the majority of women in this country, already too great, must be rapidly augmenting.

When we scrutinise the mortality of the sexes at different ages, we arrive at some definite and interesting results. That of infants, after slightly rising for a long period, has pretty decidedly fallen during the last few years, owing more, I fear, to a succession of cool summers than to the undeniably good effect of the sanitarian movement.

Here let me direct attention to a very able paper by Dr. Ransome, of Manchester, on the Death-roll in Eng-



land, wherein, refuting Mr. Baxendell, he shews the real and considerable benefit we owe to that movement.

To return. The death-rate of males between the ages of 1 and 25 has undoubtedly improved. This is a great gain, and is probably due partly to the influence of what is called factory legislation, and partly to the success of the efforts of our profession, and especially of the officers of health, in combating zymotic disease. But after 25, and more decidedly after 35, and thence very nearly to the close of life, there is an increase of the rates, not quite so large as the decrease in youth and adolescence, but still unmistakable.

In women also the infantile death-rate has somewhat increased. There is then a pretty decided decrease up to 35, a small or doubtful decrease up to 55, and then a doubtful position till the close of life.

All this is confirmed by an examination of the proportion borne by the deaths at different ages to the total deaths. The result of this examination is, that in males the deaths in infancy have considerably increased, that those from 1 to 35 have considerably lessened, those from 35 to 75 much increased, and those from 85 upwards distinctly decreased; while in females the deaths in infancy have increased, those from 1 to 35 have greatly decreased, those from 45 to 85 have decidedly increased, and those over 85 slightly diminished. This last slight decrease, occurring in a population with an increasing birth-rate, is probably apparent rather than real, but in the men, I suppose, it means that fewer of them attain extreme old age.

To recapitulate: it appears that infants are either worse tended or exposed to greater perils, that females get through youth and the child-bearing period better

than formerly, and do not break down any earlier, if so soon; but that men, though they also do better in early life than they used to do, have a tendency to break down rather earlier. This last statement is confirmed by the increase in the average amount of sickness suffered by members of friendly societies.\*

Neison some time ago affirmed the paradox, that the mortality and public health in England, taken as a whole, were not improving, though they were improving both in the towns and in the country districts of England. Though it sounds strange, it is very nearly true. For as the towns have a rate on an average about 5 per 1,000 worse than the country, and as the towns grow while the country has ceased to do so, the citizens every year bear a larger and larger proportion to the rustics, and have a greater influence in determining the average death-rate for all England. But it requires a rather nice selection of years to demonstrate the truth of the paradox.

I will now bring this discourse to a close, with a brief review of the manner in which the various causes of death have respectively waxed and waned during the last forty years.

At first sight one gets the impression that changes of this nature have been numerous and great; but closer investigation shews that most of these apparent changes are in name rather than in nature, depending on altered pathological views, on greater nicety in diagnosis, on inclination to euphemism in the certifiers, or on mere fashion.

Thus Old Age is now much seldomer found in the obituary lists, some definite lesion taking its place. The

\* Scratchley on Friendly Societies, p. 20.

numbers ascribed to dropsy gradually lessen, while those given to heart disease, to Bright's disease, and to disease of the liver grow in correspondence. Premature Birth decreases, Atrophy and Debility increase and decrease alternately: probably here there is a change of name. Cancer, Aneurism and Syphilis all steadily increase: in the first and probably also the second and third of these, improvement in diagnosis is partly if not wholly the cause of the augmentation, though there is reason for thinking that cancer really does increase somewhat. Of Diabetes we may probably say the same.

The apparent increase of Apoplexy and Paralysis, which is steady and considerable, is more difficult to account for: some few deaths may have been transferred to these heads from Sudden Death, Debility, or Convulsions, but on the whole I am disposed to believe, with Mr. Welton,\* that there has been a slight increase in fatal brain disease in males of middle age.

It is curious that in these days of minute study of cerebral pathology, the number of deaths set down simply to Insanity is on the increase. Deaths from Delirium Tremens do not increase, and Dr. Farr lays stress on this in disproof of the alleged increase of drinking in England. Dr. Ransome however has certainly shewn some reason for thinking that part of the augmentation of male mortality is due to Alcohol directly or indirectly. For my own part, I believe that Delirium Tremens is less fatal than formerly because it is more judiciously treated.

Nearly the same may be said of Cardiac Disease as of Apoplexy, though change of name from improvement in diagnosis is more credible here than there. But for some

\* Social Science Transactions for 1878, p. 527.



years past Heart Disease has ceased to gain from Dropsy in the returns ; and nevertheless its increase therein, among males of middle age especially, has been notable ; and some of us, from individual observation, believe this increase to be more than apparent.

But the most remarkable, if not important, of the alterations in the returns is under the heads of Phthisis and of Bronchitis.

When the Registration System was instituted Consumption was classed under Diseases of the Respiratory System ; an arrangement unscientific, perhaps, but not inconvenient. The number of deaths ascribed to these diseases was about 60 out of 10,000 living, and under the same arrangement it would be about the same at present. But 39 of the 60 were then ascribed to Phthisis, whereas now there would be only 22. And on the other hand Bronchitis, which then accounted for one or at most two out of the 60, has so enormously increased that it overtops Phthisis itself, slaying from 22 to 26 of 10,000 living, according to the season, and ranking as the most fatal of English diseases. Meanwhile the numbers for Asthma have greatly and those for Pneumonia slightly diminished.

It is evident that the last generation of medical men referred fatal cases of chronic bronchitis to either asthma or consumption. But this is not a complete explanation.

In other English-speaking communities the same change is going on. It does so in the Scotch towns, but there we have an increase, too, in the aggregate amount of respiratory mortality.

In Victoria Bronchitis is not nearly so common, but the returns shew an increase. In the United States it is probably really infrequent. In returns from the Continent of Europe the uncertainty of nosology and nomen-



clature comes out very distinctly; but where Bronchitis is mentioned as an important death-cause it is apparently increasing, whether phthisis be diminishing or no. This is the case, for example, at Liège and Antwerp, two cities which in their atmospheric conditions more nearly represent an English manufacturing town and seaport, respectively, than any others I can find.\*

I have little doubt that Phthisis has really diminished, especially among young adults, owing to better drainage and, perhaps, better food too, among other causes. And I believe that Bronchitis has correspondingly increased, owing chiefly to the extension of our towns, and the growing defilement of the air. Greenhow long ago shewed us† that Bronchitis was a disease of towns rather than of the country. Still, the greater part of the alteration in the figures of the Registrar-General is due to the transference from Phthisis to Bronchitis of a number of deaths of infants and children, and from Asthma and Old Age to Bronchitis of a number of deaths of old people.

Possibly you may think that I have been a little too sceptical as to supposed improvements in public health—that I have drawn the shadows in my picture a little too dark. Indeed the general aspect of affairs in England just now is so gloomy, that the dull grey atmosphere tones down the bright colours of hope. But rifts in the clouds may be found if we look for them.

I have several times quoted the mortality statistics of Victoria. In that colony I find that at every period from infancy to old age the death-rates shew an improvement

\* Between 1866 and 1873, roughly speaking, the deaths ascribed to Pulmonary Tuberculosis in Liège decreased from 43 to 38 per 10,000 living, and in Antwerp from 36 to 31. On the other hand, those set down to Bronchitis rose in Liège from 18 to 28, and in Antwerp from 31 to 36.

† In his valuable blue-book of 1858.

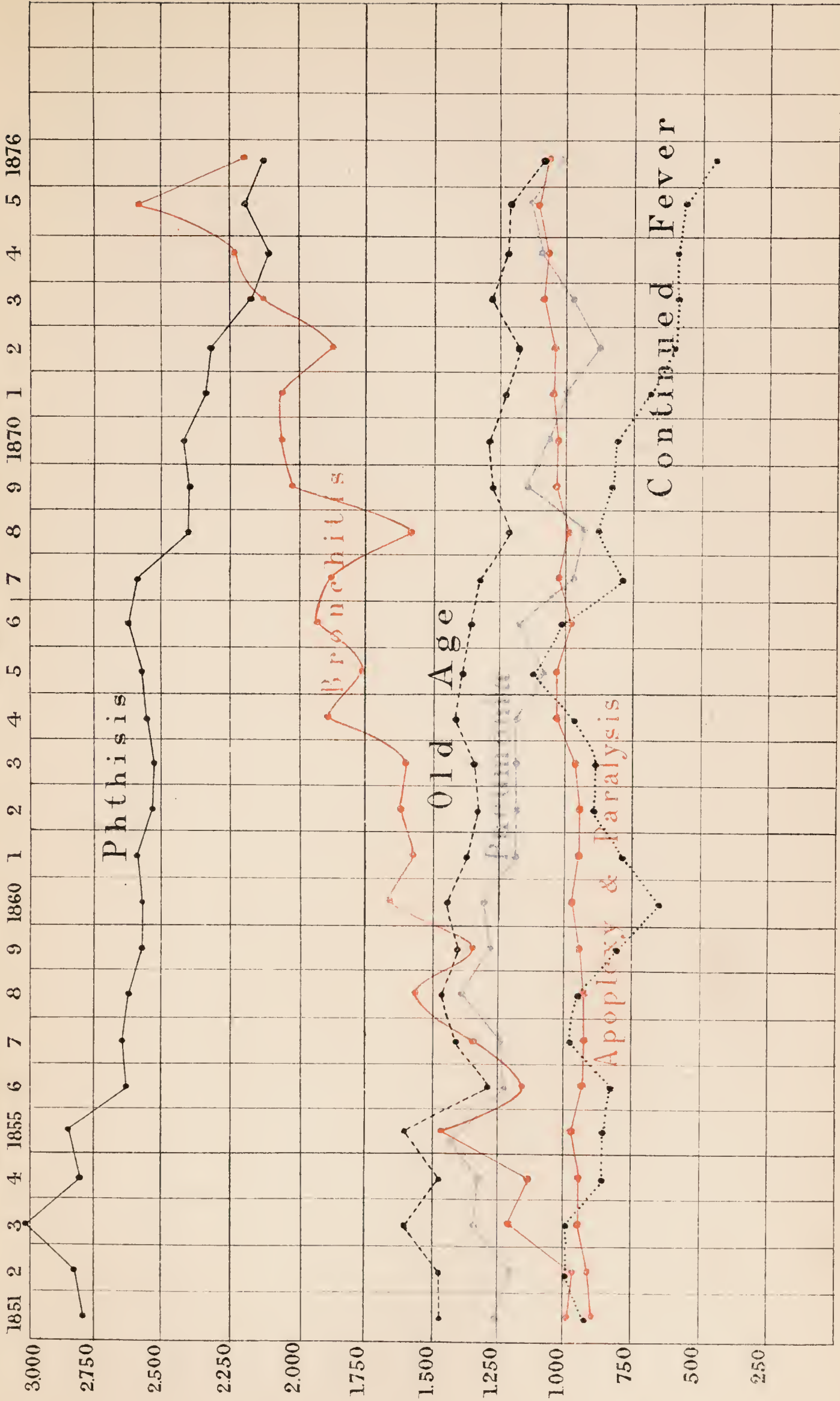
on those of the mother country.\* Nor is this altogether due to the favourable influence of a fine, warm and dry climate. For some of the lowest rates in Britain are those of the cool and moist Hebrides and Shetlands. Nor does it depend on the absence of a severe winter, for the very ideal of Dr. Farr is found in Glendale, on the bleak pastoral slopes of the Cheviots.

Many of the conditions of life that surround us are unfavourable ; but some of them may be modified, and to others we may learn how better to adapt ourselves or to offer resistance. Working with these aims we may not, indeed, live to see Hygeiopolis, but we may attain such a measure of improvement as we may be able to regard with satisfaction and thankfulness.

\* Extracted from "the Victorian Year book, 1873, by Henry Heylin Hayter, Government Statist" :—

	Number of Deaths to 1,000 living at each age.			
	Victoria, Average of 10 years.		England and Wales, Average of 30 years.	
	Males.	Females.	Males.	Females.
All Ages ...	16·68	16·13	23·33	21·51
Under 5 ...	55·08	49·66	72·42	62·46
5 to 10 ...	7·52	6·95	8·79	8·67
10 " 15 ...	3·27	3·25	4·95	5·10
15 " 25 ...	4·95	4·81	7·90	8·22
25 " 35 ...	7·85	8·28	9·93	10·15
35 " 45 ...	12·09	11·12	13·03	12·30
45 " 55 ...	17·52	13·20	18·16	15·67
55 " 65 ...	29·71	21·98	31·53	28·56
65 " 75 ...	53·79	43·10	68·54	57·52
75 ... ..	111·71	93·02	147·74	135·36

NUMBER OF DEATHS, TO 1 MILLION LIVING, FROM SEVERAL CAUSES, FROM 1851 TO 1876 IN ENGLAND & WALES.

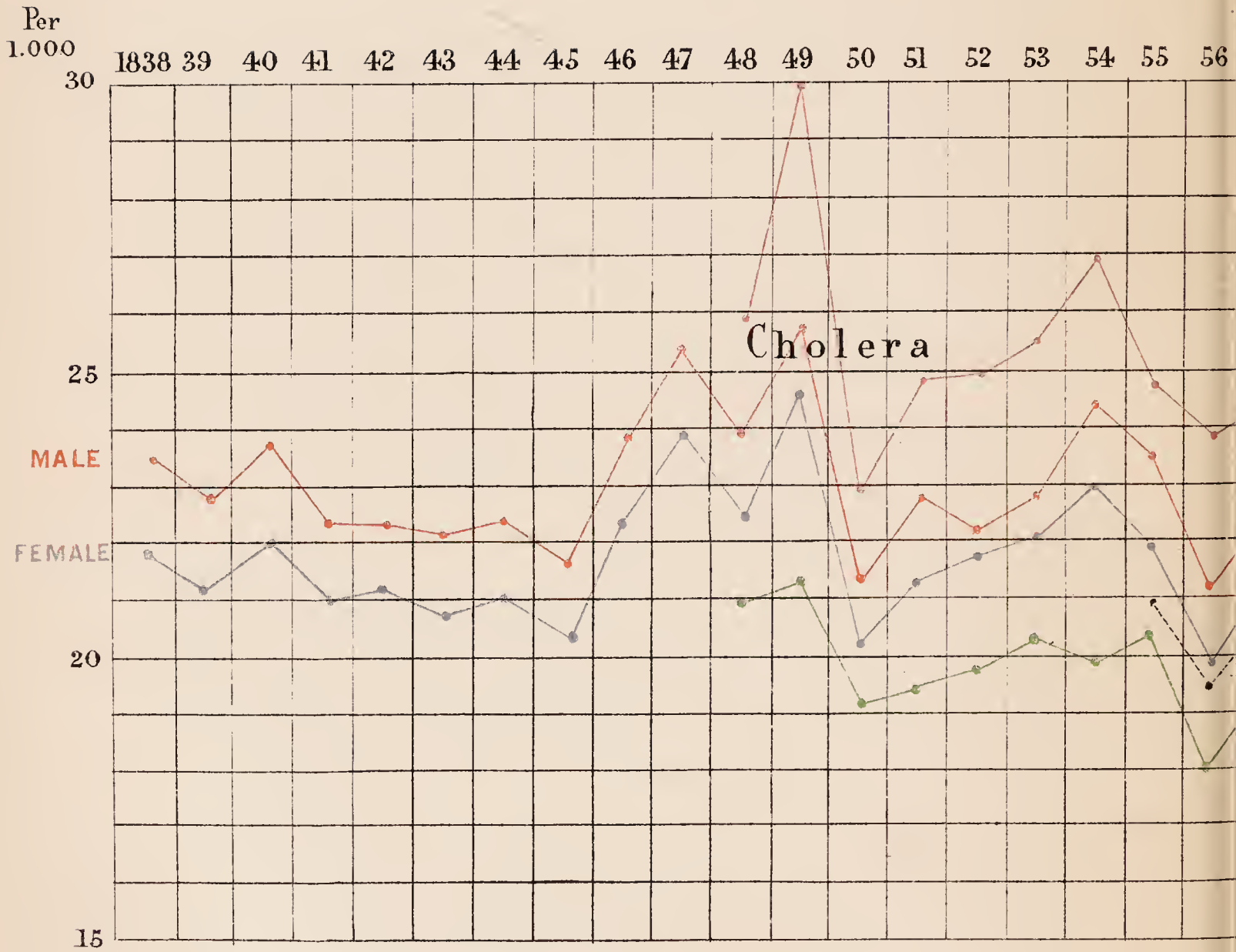




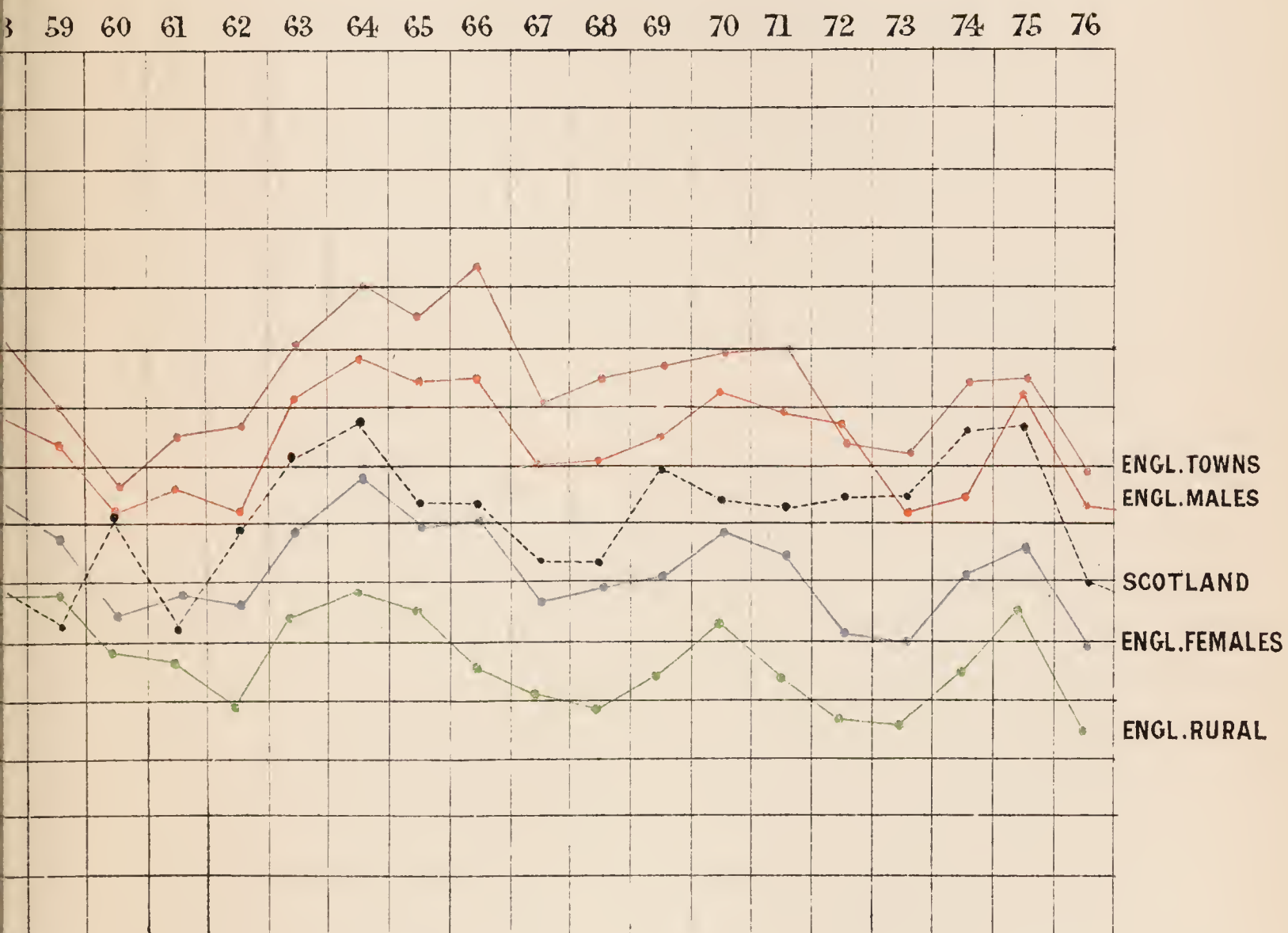




# ANNUAL RATES OF MORTALITY IN



AND & SCOTLAND, UP TO 1876.



and open communication with a very large portion of the sewer-system of Bristol. The Medical Officer of Health came to the conclusion that this last outbreak of illness in the school was one of enteric fever; and, under all the circumstances, his only wonder was that any of the inmates of the school escaped. These details shew that this last outbreak of illness in the school was connected with defects in the drains, and was presumably enteric fever.

The symptoms noted in all the cases—I quote the medical officer to the institution—were these: malaise, headache, foul tongue and gastric irritation; in some cases tenderness in the right flank and epigastrium, and in some cases diarrhœa; in all the cases fever. There were some twenty or twenty-five cases in all. Most of the boys got well under medical treatment. Four boys were more ill than the rest, and because of the severity of these cases, and because they were considered to be bad cases of enteric fever, the boys were sent to the Bristol Infirmary. Three of these boys came under my care, the other was under the care of a colleague; but owing to the courtesy of my colleague, who kindly placed his case at my disposal, I am able to speak of all four cases as if all had been my own. In these four cases sent to the Infirmary the symptoms began in the same way as in the other slighter cases and were similar, but the temperature from the first was higher and all the symptoms were more severe. Three of the four boys had been ill a fortnight before their admission to the Infirmary; the other had been ill for five or six days, and had become delirious on the fourth day of his illness. Two of the boys were fourteen years of age; the others were twelve and thirteen respectively.



Such is the history of the outbreak of illness in the school and of the four boys up to the time of their admission to the Infirmary. I need not describe in detail the symptoms and progress of each case from this point; any one case is, in its main outlines, a repetition of the others. Nor need I recite the details of any one case, for it will be enough if I describe what general features all four cases had in common.

First, the early and continuing symptoms were such as might belong, and often do belong, to enteric fever. Pyrexia—a temperature not remarkably high, but with marked diurnal exacerbations and remissions; a pulse-rate from 100 to 128; dry and coated tongue; tenderness in the right iliac fossa; in two of the cases flatulent distension of the abdomen; apathy and drowsiness, alternating with delirium (chiefly talking and moaning), and passing finally into coma a longer or shorter time before death; in one case early diarrhœa; in another case the only motion passed whilst in the Infirmary had the characteristic look of a typhoid stool.

Secondly, the subsequently developed symptoms were such as might belong, and often do belong, to acute tubercular meningitis.\* The “*tache cerebrale*” was very marked in all the cases; there was marked hyperæsthesia everywhere; the patients were in a listless, apathetic state, lying with knees flexed on the abdomen,

\* No doubt the thought will occur that I make an unnecessary distinction here, for we all know how often, at the age of these boys, the symptoms of enteric fever simulate those of tubercular meningitis, *et vice versa*, how impossible it often is to diagnose with confidence which of the two conditions may be before us. It is just this difficulty that recurred in these cases, and it is because of the difficulty that I make the distinction—an arbitrary distinction, I confess. Perhaps, but for strong presumptive evidence in favour of enteric fever, these cases might have been set down without any misgiving as cases of tubercular meningitis. But in trying to realise this, one is only thrown back into the old difficulty once more.

crying, as if in pain, when required to move, sleeping in snatches and very peevish when roused. Then, in two cases, a day or two before death, ptosis of the right eyelid and dilatation of the right pupil were observed. In one of these two cases a solitary attack of left-sided convulsions occurred about a week before death. Severe headache was always complained of while the patients were sensible, and afterwards the restless movements to and fro of the head, and the gestures, bespoke its continuance. But all these cerebral symptoms were mixed up with those of the other group; if anything, the clinical features—in three out of the four cases, certainly—were more expressive of enteric fever than of meningitis.

To this account of symptoms I may add some symptoms referrible to the lungs. There were cough without expectoration, harsh and somewhat rapid breathing, localised and irregularly scattered dulness on percussion. Not all the boys shewed these symptoms, nor were the symptoms so combined or marked as to serve any useful or diagnostic purpose.

So then, I think, this history of these four cases leads to the conclusions that the boys, poisoned and fevered by sewer emanations, had symptoms indicative of enteric fever or fever of the typhoid mode, and that they developed tuberculosis during the course of their illness.

All four boys died. Next, then, for the morbid appearances. And in giving these I shall make it the occasion to supplement and complete the clinical history already given; I shall refer to particular symptoms or particular differences in symptoms in connection with the *post-mortem* appearances as I go along.\*

The arachnoid and pia mater at the base of the brain

\* The *post-mortem* appearances are given in a tabular form at the end of this paper.

was thickened and opaque, and the sides of the sylvian fissures were adherent, in all the cases. In one case, the thickening and opacity were tolerably universal over the whole brain; in another case, two small arborescent patches of lymph were found on the surface of the parietal convolution of the left hemisphere. Tubercles were seen—in some cases many, in others few—where the thickening and opacity were. The ventricles contained more or less of excess of liquid, and the choroid plexuses were studded with tubercles. The case in which the usual symptoms of meningitis were most marked had fewest tubercles and these least developed, and had least liquid in the ventricles and least adhesions. If this case had been a sporadic case, so to speak, and if the history of exposure to sewer emanations had been absent, it might no doubt have been put down from the symptoms as a case of tubercular meningitis. Yet there was more of tubercular meningitis in the other cases than in this one. And in the case that, from the symptoms, was most typhoid and least tubercular, the anatomical signs of tubercular meningitis were the most marked; in this case small and incipient tubercles were found in the course of nearly all the small vessels of the pia mater, in the sylvian fissure, and elsewhere. In all four cases, then, tubercles were found in the brain; and the consequences of tubercle, yet not greatly advanced, in thickening and opacity of the arachnoid membrane and pia mater, and in effusion of liquid into the ventricles—more or less.

Tubercles were found scattered over the pleural surfaces of one or both lungs in three out of the four cases. In one of the three—the one in which life had been most prolonged—there were recent pleural adhesions on the



right side, and there was a caseous mass about an inch in diameter at the apex of the right lung. In the boy whose pleuræ were wholly free from tubercle, the symptoms had been pretty equally indicative of enteric fever and tuberculosis; this was the boy who had patches of lymph in the brain and who passed the characteristic typhoid stool. Two cases had tubercle scattered through the tissue of both lungs; in one of these cases both pleuræ were tuberculous, in the other both pleuræ were free from tubercle. In the other two cases one lung only and one pleura only was affected; the left lung and the left pleura in one of the cases, the right lung and the right pleura in the other—this right lung had the caseous mass. *Caseous bronchial glands were found in all the cases.* The situation of the cheesy glands in relation to the disposition of the tubercles in the lungs and pleuræ is interesting. In one case, as I have said, both lungs were tuberculous, but both pleuræ were free of tubercles; here the bronchial glands on both sides were caseous. In another case, both lungs and both pleuræ had tubercles, though these were few and scattered; here the bronchial glands on the left side only were caseous. In the remaining two cases, one lung only with its corresponding pleura was affected; here the caseous bronchial glands were on the same side as the affected lung and pleura.

The mesenteric glands were not caseous, but enlarged and indurated in all the cases.

The Liver, Spleen and Kidneys were affected with tubercle, more or less, in three cases; in one case only were tubercles found in the substance of the Liver. Ulceration of the intestines, chiefly of the ileum, and most marked at the ileo-cæcal valve, was found in all four cases. I will specify the intestinal lesions found in each case.



In Case 1.\*—The intestines, large and small, were hyperæmic in patches, almost in their whole length. Peyer's patches were congested and enlarged almost throughout the area of their distribution; those at the lower end of the ileum and at the valve were extensively ulcerated. The rectum contained compact yellow fæces, and the small intestines contained liquid and shreddy material which was in places adherent to Peyer's glands. There was no tubercle in the intestines or peritoneum. This case was examined at the end of the third week of the illness.

In Case 2.—The ileum shewed several sharp-edged ulcers, occurring in patches, down to and including the valve. The large intestine was congested. The intestines contained yellow fæcal matter similar to that found in case 1. The duodenum was studded with tubercles in a great portion of its length. This case was examined in the middle of the fourth week of the illness.

In Case 3.—The ileum was congested throughout. For a distance of two feet from the ileo-cæcal valve upwards, and also at the valve, Peyer's patches were ulcerated. The cæcum was congested and contained ochre-yellow fæcal matter. No tubercle was found. This case was examined at the end of the third week of the illness.

In Case 4.—The intestines, both large and small, were ulcerated. The ulcers appeared as if clean-cut, and their bases were thickened; they were most numerous at the ileocæcal valve, but extended upwards to the duodenum and downwards along the cæcum. The peritoneum was in parts affected with tubercle. This case was examined at the end of the second week of the illness.

An interesting enquiry meets us here. Were these

\* The numbers here correspond with the numbers in the table at the end of the paper.

intestinal ulcers tuberculous or typhoid? We are familiar enough with ulceration of the intestines in phthisis, and ulceration of the intestines also occurs with acute intestinal tuberculosis. But here there was certainly no phthisis, and only in one case were any tubercles found in the intestines; in this case (case 2) where tubercle was found there was no ulceration, and where ulceration was found there was no tubercle. It is hard to believe that these ulcers were formed at the sites where tubercles had grown and fused and softened and wrought necrosis of neighbouring tissues. In some of the cases the long diameter of the ulcers was at one place axial and at another place transverse in the same case. The edges of the ulcers were never ragged and sinuous, but thickened and sharp; and the floor was never nodular, but smooth and sloughy. If it be allowed that extensive ulceration of the intestines may occur in tuberculosis before or independently of any eruption of tubercles in the walls of the intestines, then we may regard the ulceration in these cases as tuberculous. If there was anything in the characters of these ulcers that clearly marked and identified them as tuberculous, then we may allow that tubercles had formed where, and only exactly where, the ulcers were found. An appeal to the shape and other characters of the ulcers cannot, I think, go for much in deciding upon their typhoid or tubercular nature. We need only go into any museum containing specimens of intestinal ulceration produced by different diseases to find that such a hard and fast distinction is in fact, whatever it may be in theory, unsound. It is impossible to decide the question by an appeal to morbid specimens independent of theory, and modern views on tuberculosis forbid the theories that, in regard to this question, do duty in

most of the text-books for facts. Leube\* has said upon this question:—"The only way in which we can be absolutely certain in regard to the tubercular character of intestinal ulcers is to include under this head only those ulcers in whose immediate vicinity miliary tubercles can be detected." The ulcers in no one of my cases will answer to this test; they were without doubt the ulcers naturally belonging to typhoid fever.

Such is the history of these cases from first to last. Let me briefly recapitulate the points in the history, and present them in a form convenient for my further purpose.

An epidemic illness occurred in the school where the four boys lived, and its origin was traced to defects in the drains within the house. The illness was marked by symptoms which could leave no reasonable doubt of its specific or typhoid nature. The four boys had the fever badly from the first. After they had been fevered for a longer or shorter time—three of them for nearly a fortnight and the fourth for a week—symptoms of tubercular meningitis arose. On examination after death, it was found that all the boys had cheesy bronchial glands, and one of them had, besides, a cheesy mass in one lung. The intestines, chiefly the ileum, were extensively ulcerated, and in all the cases Peyer's glands were involved. Tubercles were found in the duodenum in one case, in the peritoneum in another case, in the lungs in every case, in the pleura in three cases, and in the meninges of the brain, along with the anatomical results of inflammation, in all four cases. All these boys had the same constitutional stamp and were strumous, for they all had cheesy bronchial glands; they all lived under the same conditions, and were subject to the same influences whether

\* *Ziemssen's Cyclopædia of Medicine*, Vol. VII., p. 406.



for good or for evil; they all were exposed about the same time to infection from sewer poisons, and all got typhoid fever in consequence of the exposure; after the fever had declared itself, they all were taken to the Infirmary, and there placed under the same conditions; and there they all developed, in a similar way, tubercular meningitis with more or less of general tuberculosis according to the duration of life.

Now, when I watched these cases, and saw the *post-mortem* appearances, and when afterwards I thought upon all the facts and circumstances of the histories, one question seemed to press itself and repeat itself with peculiar force upon my mind. The question, shortly put, was this: Had the fever anything to do with the development of tuberculosis in these strumous (or caseous) boys? It is my purpose, in what follows, to discover what answer to this question the facts of my cases and the circumstances of our present knowledge may allow. And whether or not an answer in the affirmative should come out of the enquiry, some additional light may be thrown by means of it upon a not too well illuminated corner of pathology.

This purpose requires, in the first place, that we should endeavour to arrange the various facts, bearing on the question and common to the cases, in that order of sequence and relationship which may naturally belong to them. The things that concern us most, as bearing on the question and as common to the cases, are three—caseous matter, fever and tubercles. These are, however, no more in themselves than central facts; each as a centre attracts around it other facts whose relations to the centre will not be in dispute. Thus, caseous matter represents the process of caseation and what belongs to



the strumous state ; so, fever represents the known facts belonging to the febrile process in general and what relates to the special fever of these cases in particular. But it will be convenient and useful, and withal strictly just, to keep these three central facts present to the mind and uppermost in the complicated enquiry which is before us.

And first, as to an order of sequence and relationship in time ; the *chronological order* of the facts. We can say, without misgiving, of certain morbid appearances in a given case that they are newer or older than others in the same case. If we find Peyer's glands here congested and there ulcerated in the same intestine, we have no difficulty in assigning to each condition its relative age. When caseous glands and tubercles are found at the same time in the same body, the caseous masses must, we know, be older than the tubercles, and the tubercles must be later in their appearance than the caseous matter—without regard to any other relations they may bear. Caseation is, as Rindfleisch says, “the anatomical criterion of chronicity as regards inflammation ;” it is, in itself, the sequel to a series of prior and long-enduring inflammatory and degenerative changes. So there is an age for tubercles, although we cannot tell with certainty their absolute age. Some tubercles are older than others in the same case (we can, it is said, distinguish incipient tubercles from formed yet recent tubercles) ; and tubercles, again, are older than arachnoid opacity or lymph when these are found in the immediate vicinity of tubercles and are fresh.\* We have enough knowledge

\* The re-sorption doctrine has so dominated in this region of pathology during recent years that the facts bearing on the chronological relations between inflammatory processes and tubercles in their neighbourhood have been collected and presented so as to square with this particular doctrine. The whole question is beyond our present limits. What has been stated in the text is sufficient for our purpose and is independent of speculation.

about these things to enable us to place the facts of these cases, and the processes they represent, in this their order of sequence in time:—The caseous masses were the oldest and first-formed anatomical appearances; they represent strumous processes arising and going on and brought to a conclusion before the illness to which the four boys succumbed. Fever next arose, after a lapse of time (and of how much time we cannot tell), fever induced by poisoning with sewer emanations. The intestinal ulceration followed upon the onset of the fever; this ulceration tells us of the nature of the fever and confirms the etiological and clinical evidence for its typhoid character. The miliary tubercles in the lungs and pleura, in the arterioles and meninges of the brain, and in other parts were nearly of the same age, and they succeeded the onset of the fever by nearly two weeks. And, finally, as the last events in the series, come the consequences of the eruption of tubercles in the brain—the meningeal inflammation with its effusion and lymph.

Next, comes the arrangement of the facts and what they represent in a *consequential order*; in the natural relations they bear in the order of cause and effect.

The fever, no doubt, caused the intestinal ulceration; at least, that part of the fever which is specific. There is no ground for supposing that the specific part of typhoid fever, or of any other fever, has any special causative relations with tubercles. A few cases have been recorded in which tuberculosis occurred a longer or shorter time after typhoid fever, and tuberculosis occurs commonly after measles and scarlatina. But it has never been pretended that these fevers had, by what is specific to them, any share in the tuberculosis; nor would such a pretension have any weight, even in cases where

(as in my cases) tuberculosis developed in the course of a specific fever. The specificity in a fever consists in certain special circumstances of cause and local lesions. These are the things which, superadded to what is common to all fevers, make *a* fever and give a specific character to it; and the things superadded in one specific fever are different from those superadded in any other. If we could shew that the things specific to any one fever were essential to, or immediately concerned with, the development of tubercles, we should thereby effectually shut out all other specific fevers from any share in a like result. The things specific to any one fever belong to that fever and to nothing else. If, for instance, we should be disposed to connect the development of tubercles, during or after typhoid fever, with the circumstance of the specific typhoid poison having a special effect upon the lymphatic organs and being specially concerned with these as regards its phases of developmental change (Liebermeister), what shall we say of measles, during or after which specific fever tuberculosis is much more frequent, and in which disease the special circumstances of cause and local lesions are quite distinct from those we find in typhoid fever? If tuberculosis occurred with typhoid fever on account of the specific characters of this particular fever, then tuberculosis could not occur with measles on account of the special characters of the fever in that exanthem, and so for any fever in which special circumstances of cause and local lesions mark it off as a specific fever. If we have reason to believe that more than one specific fever has intimate consequential relations with tuberculosis, we must look for the relations in the things common to the fevers. We cannot suppose that the fever which affected our four boys was, on



account of its specific characters, of a kind to induce their tuberculosis. It is with fever as fever, with the essence of all fevers, and with that which is proper and common to them all that we have now to do. The specificity is, indeed, of this use to us here. It enables us to say that the fever observed in these cases at the beginning of the illness did not belong to, as being dependent on, the tuberculosis; that there was fever, quite apart from the tuberculosis, continuing throughout the illness. It enables us to say that fever was present earlier than and independently of any fever that might have necessarily arisen out of or succeeded the eruption of tubercles. Leaving out, then, the specific element in the fever of our cases, we find that what is of the essence of all fevers—viz., pyrexia and what belongs to it as such, preceded the tuberculosis and went on side by side with the development of tubercles in different organs and parts; and this in all the cases, and in cases also otherwise similar in their circumstances and events.

In uncomplicated tuberculosis—that is, in cases where there is no accompanying specific disease nor anything going on in the body, besides the general eruption of tubercles, with which any co-existing fever could be connected—there is fever; and where the tuberculosis is very general and the case is quite uncomplicated, fever of a high degree. If we take a typical case of acute miliary tuberculosis—one which is characterised anatomically by the presence of disseminated grey tubercles in the lungs, with grey tubercles also in the peritonæum, liver, spleen, kidneys and pia mater of the base of the brain—we find that the beginning and the course and the end of such a case is marked by fever. Such a case, when uncomplicated, commonly begins with shiverings, frequency of



pulse, constitutional disturbance and elevation of temperature—often to a high degree. The disease may, at its outset, be mistaken for intermittent fever; it might be mistaken for extensive bronchial catarrh, accompanied by fever, but for the violence of the fever, the absence of physical signs and the rapid collapse; it may easily be mistaken for typhoid fever. The patient is killed by the fever, just as he might be consumed by the fever in uncomplicated typhoid disease; one who has died of acute miliary tuberculosis has certainly died of an acute febrile disease. In our treatment, too, fever is the most important indication. Now, there is no evidence that tubercles, the grey miliary granulations, do come into existence and grow for a while, in a latent sort of way, without fever of any sort or degree. On the other hand, there is no evidence that fever is an early and essential and integrant part of tubercle-formation in the anatomical sense. We are quite as much entitled to say that the fever invariably observed to accompany acute general tuberculosis in its uncomplicated forms is, in part at least, extrinsic as we are to say that it is wholly intrinsic.\*

We can, of course, speak with some certainty of the fever occasioned by tubercles—that fever due to consequent inflammatory disturbance. We can certainly say that any fever due to the cerebral inflammatory disturbance in our cases was intrinsic; but then it was later than and superadded to the other and earlier fever. With such fever, however, we have no concern.

The fever, as noted in these cases, was of a high

\* The point here briefly raised cannot be worked out as an argument within the limits of this article. It must suffice to indicate its bearings on the question under discussion.

degree at the first. We know that in those boys of the school who had fever more lightly—in whom the pyrexia was much less intense—no tuberculosis occurred. Yet, presumably, many of those boys who had slight fever and developed no tubercles were constitutionally like our four boys. The medical man who had attended at this school for many years told me this fact: he had noted that when the boys of this school got enteric or other febrile diseases, and “it went hard with them,” they almost invariably had tubercles when examined *post-mortem*. And from my own experience, during nearly eight years past, of many boys who have come from this school to the Infirmary with febrile diseases of various kinds I am able to corroborate the fact. There is no reason to suppose that the four boys would have become tuberculous at the particular time if it had not been for the accident of exposure to typhoid infection and the consequent advent of fever. Fever and tubercles, then, come into such close alliance in these and other cases as to give reasonable ground for a belief in some consequential relations between the two. What these relations may have been we shall enquire further on.

The caseous matter and all that it represents stands first in the chronological order. But it stands, apparently, alone and apart from the other things; for, although all four boys had cheesy bronchial glands at the time when fever and tubercles arose, the process by which the glands came into this state began and ended and had passed, leaving only its anatomical result—caseous matter, before the onset of the fever, which is the next succeeding and delayed event in the chronological order. “In the very great majority of cases of tuberculosis of the pia mater (and of the whole body) a primary

focus of yellow cheesy degeneration is to be found.”\* The frequency of the concurrence of caseous matter with tubercles in the same cases has become so well established that some even prefer to accept the probability of the cheesy focus being in some unusual place or overlooked rather than to accept its unequivocal absence. We may allow the very frequent concurrence of caseous matter and tubercles to be a well-established fact. The caseous form of degeneration is a characteristic of scrofula. “Tuberculosis is built up almost without exception upon scrofula as a basis. Pathological anatomy shews it to be characteristic of scrofulously diseased assimilation that almost all inflammatory processes shew no inclination to absorption nor to an active formation of pus, but, on the contrary, a tendency to retrogressive metamorphosis, which, to the naked eye, offers the appearance of cheesy degeneration.” “Experience in thousands of cases proves that in the far greater majority it is scrofulous individuals, or those who have been scrofulous, who are attacked [with tuberculosis]. It is not necessary that the individual should have the outward marks of scrofula; this, however, is necessary, that in him should exist that tendency to bring inflammatory exudations to a cheesy degeneration. It is, then, easily understood that all sorts of acute diseases which cause a great number of inflammatory lesions lead, in the case of individuals with a disposition to scrofula, to dead cheesy exudations, instead of leading to such as simply suppurate, or become indurated or are absorbed.”†

Whatever other conditions or circumstances may be

\* *Huguenin—Ziemssen's Cyclopædia of Medicine*, Vol. XII., p. 512. Many other authorities might be quoted to the same effect.

† *Huguenin—Ziemssen's Cyclopædia of Medicine*, Vol. XII., pp. 519, 521.



required for the effect, there can be little doubt that caseous matter, or its basis scrofula, has, in the great majority of cases, some consequential alliance with the tubercles in acute miliary tuberculosis. On the ground, if not of absolute uniformity of concurrence and succession, yet of an approach very near to this, we may feel much confidence in the existence of such natural relationship.\*

We have already shewn grounds for the belief that the fever had some consequential relations with the tubercles.

We have still to enquire if the fever may have had any consequential relations with the caseous matter or scrofula. Do these hold in the consequential series the same position they hold in the chronological series? Caseous matter or scrofula and fever may form an aggregate (not necessarily the entire aggregate) of conditions which are concerned in the production of the effect—tuberculosis. Fever may be one of the essential circumstances under which caseous matter begets tubercles, or under which the scrofulous become tubercular; it may be a link in the chain which, when all the conditions have been fully enumerated, will be a complete statement of the cause of tuberculosis. There are facts in my cases supporting this relationship. Fever comes in between caseous matter or scrofula and tuberculosis. As a matter of fact, tubercle did not appear until the advent of fever—the extrinsic fever; although both caseous matter and scrofula had been for a long time present. It is a fact in the medical history of the school that tubercles appear in strumous subjects when fever comes. And with slighter

\* I have quoted from Huguenin; but have quoted only his facts. It is quite another question what the consequential relations may be and how they may be explained. The two questions are not usually kept apart with that separateness which explanation of natural phenomena requires.



fever than our four boys had, other strumous boys in the same school did not get tubercles. The facts of my cases and the considerations already adduced, not only assign to fever an intermediate place, but suggest for it a rôle of active intervention between caseous matter or scrofula on the one hand and miliary tubercles on the other. There would appear, then, to be fair and reasonable grounds for the inference that caseous matter or scrofula and fever and tubercles do hold consequential relations in these cases. The conclusion seems hardly to be resisted that these boys, carrying about with them caseous matter, the result and expression of struma, always ran the risk of becoming tuberculous; that it needed only the advent of fever to convert their risk into destiny. And this is as much as to say that we have ground for the belief that fever had something to do with the development of tubercles in these caseous (strumous) boys.

My purpose requires, in the next place, that we should endeavour to bear out and explain the relations already assigned. I pass on, then, to enquire how fever might be an active intervening agent; how it might be allied with caseous matter or scrofula and tubercles in consequential relations; what the fever may have to do with the caseous matter or scrofula and the tubercles; whether its relations were with the caseous matter or the scrofula.

There is a doctrine in modern pathology which professes to account for and explain the concurrence and consequential relations of caseous matter and tubercles. It is a doctrine taught extensively, more especially in Germany, at the present time, and for this reason alone we could not ignore it here. But the doctrine seems likely to fit in with our facts; it seems, at first sight, able

to give us just what we want, and to want just what we have to give. And if the doctrine will fit in with our facts, we shall speedily arrive at an answer in the affirmative to our question, whilst the doctrine itself will gain no little support.

Twenty-two years ago (and seven years before Villemin's experiments on artificial tuberculosis) Buhl first promulgated the doctrine. He concluded that "acute miliary tuberculosis was a disease arising from the re-sorption of a specific infective matter from old caseous products. These views have been accepted, with only certain modifications as regards the specific nature of the matter absorbed and the mechanism of its absorption, by most authorities since Buhl's time." Additional observations have been adduced in its favour by workers in this country and on the Continent. "So that we know that tuberculosis does arise in the human subject as a secondary illness, through the influence of caseous diseased products upon the system (Niemeyer)." \*

Huguenin† thus states the doctrine:—"The introduction of caseous detritus into the circulation of an individual causes the development of miliary tubercles. The most minute portion of caseous detritus must be looked upon as an infecting agent, as a poison, which, by direct contact with the endothelial structures, incites them to the production of miliary tubercles. The introduction of the poison into the canal system of the body takes place from within, whenever a cheesy focus exists. *The mode of infection is not yet known with certainty; it may probably occur in a number of ways.*"

Ruelhe‡ says:—"Acute miliary tuberculosis must now

\* *British Medical Journal*, Vol. I., 1875, p. 470.

† *Ziemssen's Cyclopædia of Medicine*, Vol. XII., p. 492.

‡ *Ibid*, Vol. V., pp. 616, 617.

be considered as an infective disease which may occur *whenever an opportunity arises for the absorption of caseous matter by the vessels.*" "We know of but one cause for the formation of the miliary tubercle—viz., the absorption of caseous matter. *How this produces tubercle requires still further investigation.*" He goes on to admit considerable difficulties as to the transport of the infective matter to the tubercular sites; and in regard to general infection, an acute miliary tuberculosis, he says: "probably, *after a while*, some of the matter passes into the blood-vessels; *but whether previous physical or also chemical changes, or both together, are necessary for this result is still undetermined.*"\*

The doctrine professedly gives us an infecting agent in caseous matter; it gives us diffusion, by means of vessels, of this poisonous material; and it gives us miliary tubercles as a consequence—in the places where miliary tubercles are found. The doctrine confessedly wants opportunity and conditions for the absorption and transport of the infective matter; it wants the circumstances which determine the particular effect of the infective matter on the structures which develop the tubercles.

Now, it may seem most reasonable to take from the doctrine its infective agent, its fact of diffusion by means

\* It seems necessary thus to recall the essential points in the doctrine, in consequence of the confusion which some modern modifications (so-called) have introduced. The doctrine has been much overlaid and obscured by some of these modifications, especially in connection with the pathology of pulmonary phthisis. It may be said that the doctrine now includes many other things besides caseous matter as competent to produce tubercles. But infective material includes caseous matter, and this is, in the great majority of cases, the particular form of infective material found with tubercles after death. If it is the fact that any simple inflammatory stimulus may generate the poisonous element within the body, it is still the fact that the inflammatory stimulus which is scrofulous, and leads to caseation, is by far the most frequently occurring in those who become tuberculous.



of the vessels, and its result; and it may seem reasonable to offer fever as likely to supply the missing links. Fever may, perhaps, furnish the opportunity and the conditions and the determining circumstances acknowledged as beyond the present compass of the doctrine.

Let me define more clearly what the doctrine wants, that we may see more clearly what is required of fever in order to constitute it a connecting link. Whatever may be the grounds upon which a causal relationship between caseous matter and miliary tubercles is claimed, the conditions and circumstances which bring about at the right time—the time when tubercles arise—the influence of the infecting thing upon the structures responding to the influence are still required in order to the unity and completeness of the doctrine. What the doctrine wants, provisionally if that only is possible, is some explanation of the fact that the influence is sometimes brought about quickly and sometimes is delayed. Infective matter rests awhile, often a long while, before it is succeeded by miliary tubercles—the opportunity is not always at hand. There is an interval—be it short or be it long—between the development of infective qualities in the source and the subsequent development of tubercles, and this interval has to be accounted for. Something wherewith to forge a connecting chain or to build a connecting bridge, of this length here and of that length there, is the present need of the doctrine. If we could bring the process which originally begot infective qualities in the caseous matter (for the doctrine does not require that the infective influence should be infused into caseous matter *after* the process which led to it had ceased) and the eruption of tubercles near enough together in cases where they both occur, connecting or determining conditions would,



no doubt, be superfluous. But as a matter of fact—and markedly in our cases—there is an interval. And, generally, the fact has to be accounted for that the creation of an infective focus is, in one set of cases, more or less closely followed by tubercles, and is, in another set of cases, not at any time followed by tubercles—at least, for an indefinite time, which means, practically, that the infective matter is inert. Allowing the fullest weight to the grounds supplied to the doctrine by the analogy between tuberculosis *artificially* produced in *animals* and tuberculosis *naturally* occurring in *man*, there is still to be defined what are the conditions or circumstances which bring the cause and its effect into operative contact at the right time; which prolong the interval in some cases, annihilate it (if so it pleases) in other cases, and regulate it in all.\*

If we give to the doctrine anything wherewith to span the interval, to provide the opportunity, to consummate tuberculation at the right time—the time when tubercles appear—this that we give may satisfy the requirements of the doctrine if, coming when tubercles arise, it

\* I do not know that anyone has attempted an explanation of this interval. Rindfleisch, indeed, speaks of an interval and accounts for it thus:—"We all know for how many years a scrofulous [cheesy] gland may remain as the only trace of youthful scrofula. It is eminently proper to adopt Heuter's plan, and to extirpate such a gland, in order to prevent the gradual infection of the whole body with tubercles from it." And he accounts for this delay of many years by supposing "that swelling and obstruction of the lymphatics may cause the disease [tuberculosis] to remain for a time localised in the glands." But Rindfleisch here speaks of an interval between tubercle and tubercle, between his secondary tuberculosis in one or more glands and his tertiary general tuberculosis, which last may involve all parts of the body; and he is making a doctrine for himself based on an analogy supplied by events in the invasion of malignant tumours. To bring him *en rapport* with the point now raised, we ought probably to go back to his primary inflammation, which, as he expressly says, is not tubercular. Then the interval is still more marked, and the need for intervening and determining conditions is still more apparent.

brings with it conditions for absorption and transport at least ; or if, coming when tubercles arise, it brings with it conditions for closer relations than those of mere contact between the infective material and the tubercle-producing structures at least. If fever, coming on the scene just before tubercles arise, can bring with it and supply conditions for the absorption of caseous matter from the glands and for transport of it to the tubercular sites ; then we may suppose that, once at the appropriate sites, the infective material needed no other conditions than such as it carried with it (in virtue of its infective or quasi-specific qualities) to work the result tubercle. Thus, in order that it may provide the opportunity in this sense, fever must be required to shew itself competent to bring about the absorption and transfer. But if fever fails in this capacity ? If fever, coming on the scene just before tubercles arise, can bring with it and supply conditions for a reaction between the tubercle-producing structures and the infective matter ; then we may suppose that absorption and transport were brought about by some other agency than fever, or that there never was any hindrance to the circulation everywhere in the body of that upon which the infective influence depends, so that the infective matter was already at the appropriate sites when the right time for more intimate relations came. Thus, in order that it may provide the opportunity in this sense, fever must be required to shew itself competent to bring about an interaction between the things already in contact and only waiting for conditions to work their joint result. The opportunity resolves itself into the alternative.

Can fever furnish the opportunity in the one sense or in the other ? An answer to the question depends on

what we know about the mode and circumstances of fever action. And first, as to absorption and transport.

The tissues about bloodvessels and lymphatics are peculiarly the *nidus* of tubercle. Bloodvessels and lymphatics are the channels through which whatever may be infective or irritating, or occasioning of tubercular growth, must be carried and distributed. Lymphatic glands, which are especially liable to become caseous, and which were found in our cases to be caseous, lie in the direct course of circulating and everywhere-distributed blood and lymph. These vessels must also be the channels of carriage and distribution of fever-altered blood and lymph. And both blood and lymph must be altered in fever if either is altered. If the blood and lymph do become so altered in fever as to work any physical or chemical change in caseous matter, and in this or any other way to let loose infective particles or matter, then we might expect the infective influence to seek diffusion through its natural and always ready channels of diffusion, and so be brought into the required relations with tubercle-producing tissues. If we enquire what alterations fever may produce in blood and lymph so that a loosening and absorption of infective matter may be brought about, we must admit that, as we know at present, fever gives us little help; it gives us no conditions in itself that can avail us here. We know of nothing as pertaining to the qualities of blood and lymph in fever that can be made use of for the purpose of shewing how the loosening and absorption could be effected. Cell activity, it is true, is great and circulation is busy in fever; febrile metabolism is a diverted rather than an increased metabolism; the activity of glandular structures, wherever found, is increased in fever; we may



fairly conclude that colourless blood-cells migrate more actively in the febrile than in the normal state. But none of these nor many other things which are true of fever can, as yet, be put into such form as to be available for our present purpose.

And then, if we did know of such changes to the blood and lymph in fever, that fever should be regarded as the possible agent by which absorption of the infective particles might be effected,—would fever-altered blood and lymph necessarily circulate through caseous glands or caseous matter wherever found? In order that caseation may take place and go on, both the blood supply and the lymph drainage must be cut off. Caseous degeneration, or *tyrosis*, is a process *sui generis*. It is not a species of hyperplasia, nor a drying-up of pus, nor a simple withering of emigrant cells and exudation and elements of tissue—aggregated by chronic inflammation—from lack of water. It is a progressive molecular death and decay, happening not only without suppuration, but in place of it; an independent mode of evolution proper to chronic inflammation; and its local origin and explanation is in the failure of vascularity in the cell-infiltrated tissue, going on parallel with a continual and great accumulation of young cells, whereby the cells and tissue alike progressively yield to the withholding of the first conditions of their life. Encapsulation of the cheesy mass and induration of surrounding tissues is not infrequent; this, in spite of the assertion that encapsuling, though a considerable obstacle, is not an absolute obstacle to re-sorption (Wagner), can hardly be regarded seriously as anything but fatal to the effect of any influence which fever can be supposed to exert. We must admit a cessation or removal of the elementary physiolo-



gical conditions of life—viz., circulation and absorption—in order that caseous foci should be formed at all. And if we should be disposed to make much of the fact that the circumstances under which absorption would have to occur in our cases are pathological circumstances, it is inconceivable that any pathology can account for the re-appearance and restoration of that integrity of structure which is a first condition of absorption, but which was effectually destroyed in the formation of the caseous foci.

But, in the neighbourhood of the caseous foci the conditions for absorption of infective matter from the foci into blood and lymph streams may still be present and more active, in the febrile state, than is wont. It may be that febrile metabolism in the immediate vicinity of the caseous foci may supply conditions for absorption. And softening processes under the influence of fever, or the influence of soaking plasma during the activity of the fever time, may yet prevail. These things are plausible, and they may be more or less probable circumstances in favour of the view that fever brought absorption in its train; and we are now concerned in the attempt to make the plausible and probable credible and feasible.

There was no evidence in the cases that any softening had taken place, even when fever had been present for a period of four weeks. Without a clear and unmistakable liquefaction we can hardly invoke the aid of the forces concerned in absorption, not even filtration. We are acquainted with no evidence for absorption of undissolved matters. A very doubtful approach to the conditions we want here might perhaps be suggested by Saviotti's migration of connective-tissue and pigment cells *into* capillary bloodvessels, if the very opposite state

of things to that of stagnant or sluggish circulation did not prevail here, and if we had not to deal with matter already dead instead of living protoplasm. In round-about ways and by far-fetched suppositions only can we find any grounds for the possibilities under present consideration. Considering the circumstances of the formation of caseous foci, we are not bound to require of fever nor suppose fever to be able to do more in regard to caseous matter than we should require of it, or suppose it able to do, in the case of any other matter under similar circumstances. Something more than fever is, at least, required, and something of whose existence we have no hint. It would seem to be necessary to assume that caseous matter has some very special disposition to get away from its nests—no doubt a disposition which would suit the doctrine well, and one which possibly, to some minds, might range under specificity—in order to make the possibilities here raised even plausible.

We find, then, no reason to believe that fever, coming when tubercles arose, did bring with it conditions for absorption and transport of caseous matter. Not only is there want of evidence on the point; the conditions for absorption and distribution of infective material from the foci were much more present and adequate at a stage preceding caseation than at any time after caseation had been once established.

This brings us to the question whether fever can furnish the opportunity in our second sense. Can fever provide for an inter-action between the things already in contact—the infective thing and the tissues which bear tubercles?

We still deal with caseous matter. From our point of view it is inconceivable that the matter was transferred

at any time after caseation in such cases as I have related, where no inflammatory or other softening and liquefying processes had been at work. Yet, if fever can shew us how a reaction takes place between caseous matter and the tubercle-bearing tissues, and if fever can bring with it conditions such as may determine the particular effect of the matter on the tissues, our view would seem to be imperfect and erroneous ; we might then admit the transfer by some other agency than fever, although under circumstances not declared and obvious.

What, then, do we know of the mode and circumstances of fever action that may enable us to answer the question ?

The state of things in fever is a state of activity. Cell-life is active and circulation is busy ; the activity of glandular structures, wherever found, is increased. We may fairly conclude that colourless blood-cells migrate more actively in the febrile than in the normal state. We know little about the effects of increased temperature upon nutritive processes that bears upon the present question, though, no doubt, increase of body heat brings about qualitative variations in metamorphosis, and metabolism is diverted rather than increased in fever. The blood and lymph are more or less charged with the products of an increased metamorphosis and imperfect oxidation. We know certainly that fever-altered blood and lymph must be irritative to living tissue, and perhaps especially so to the living tissue in which tubercle finds its conditions for growth. The circulation of half-changed albuminoid products in fever would alone make the blood and lymph irritative to living tissue ; and these products do sometimes cause, especially when their elimination is interfered with, local secondary inflammations in structures which are also the seat of tubercles.



When we take account of this state of activity, especially in cellular and glandular structures, and this irritative quality of blood and lymph which characterise fever, we seem to gain some ground for the hope that fever may prove the exciting cause of a reaction between caseous matter and tubercle-bearing tissues.

Caseous matter must have been at the tubercular sites before fever came on the scene or after the onset of the fever. We cannot suppose caseous matter to be less irritative than fever-altered blood and lymph; and, by the doctrine, it is armed with special qualities which fever-altered blood and lymph have not. Whether caseous matter came first on the scene or whether it came later, whenever it came it should have no need of another irritant in order to work a result which, by its own nature and by the doctrine, it is especially fitted to bring about. Why, then, coming earlier, did not caseous matter do what it is quite as competent to do as fever is? Or, coming later, what is the need for fever at all? If we intercalate fever we must say that caseous matter is competent to affect the tubercle-bearing tissues only to a certain extent; and we must define or give some meaning to this limited influence. We might, perhaps, assume a *role* for caseous matter and speak of it as working some preparatory changes in the tissues, changes which it required some irritative or exciting cause to complete; we might suppose that caseous matter, as it were, kindled a spark which it required some irritant, like fever, to fan into flame. When we consider the nature of caseous matter, the fact that the term infective is, apart from irritation, only an abstract idea, and the absence of all evidence for the possibility of such a series of events as is here assumed—we must reject the supposition. At least we:



are at liberty to offer our own view as to the improbability of caseous matter being transported at all as an argument against such a relationship between caseous matter and fever. We cannot even shew that caseous matter and the tubercle-bearing tissues ever were in circumstances to be brought into intimate relations, and now we find that what fever would be competent to do should have been done quite as easily, or even better, by the caseous matter itself independently of fever.

We need not, however, hold the doctrine to caseous matter as the particular infective agent. The advocates of what may be called the caseous doctrine have been forced to go back to a time beyond caseation for the origin of infective qualities in caseous matter; they require an elaboration of poisonous qualities during the inflammatory process which eventuates in caseation. At this time the matter has not become dead and cheesy, and there is certainly no hindrance to the absorption and free circulation of the infective material. And even the later modifications of the doctrine, which assign to any inflammatory products an infective power, still suppose the infective quality to be generated within the body at the time when these products are in course of formation by the morbid reaction of tissues to a simple inflammatory or other stimulus, and when there is no hindrance to absorption and circulation. On our own view, conditions for absorption are present and adequate at the time when the cells, which form so prominent a feature in the chronic inflammation that goes on to caseation, are crowding the glands; and these living and active cells are likely one would suppose, if anything is likely, to generate or to receive an infective influence and to carry it elsewhere.

From this point of view the only way in which I can

conceive the doctrine to fit in with the facts of our cases is this: We may suppose that cells, or products, or other matters not yet dead and cheesy, become recipients of infective qualities during the inflammatory or other changes to which they owe their existence; that, carriers of the evil influence, they circulate at this time; that, getting, as they could get, to the required sites—the first places after the blood-vessels where anything like stagnation occurs—they there impregnate the tissues during the changes incident to ordinary nutritive metabolism. Then, if any irritative or stimulating influence operated, at the same time or at any future time, upon the tissues which had thus become impregnated, we might suppose the tubercular growth to be the special response on the part of the infected tissues to the irritative influence. And further, this response might occur at the time of infection and circulation in the near neighbourhood or within the sphere of the inflammatory or other infective process. Or, when the advent of tubercle was delayed, or tubercle appeared later in distant parts, the response might wait for appropriate exciting circumstances, such as fever, to call it forth.

Even with tissues infected and impregnated with an influence derived from matters not yet dead nor caseous, whatever their source; and with fever as irritating and exciting because of what undoubtedly belongs to it; and with these circumstances reciprocally operative to bring about the development of tubercle—we must still ask for some explanation and definition of the abstract idea infective; we must attach some precise meaning to the term.

It would lead me too far to follow out in detail the trains of thought here suggested. There would be much



to say as to the doctrine in its later forms, starting with infective qualities at the source. The idea conveyed in the term infective and in the absorption of something in the nature of a poison or virus from inflammatory or ulcerative or other foci appears to me to be the residue of an older view, which does not gain in clearness when separated from that view and adapted to newer facts and analogies. The fault of the later views appears to me to consist in this, that facts have been assimilated with portions and remnants of former hypotheses rather than with one another; that, instead of new departures, reconciliations between things difficult or incapable of reconciliation have been attempted. Analogy, in particular, has been overstrained. In the case of malignant tumours and miliary carcinosis (much relied upon for analogical proof of the later doctrine) the evidence for causal relationship between primary sources and secondary effects is wholly anatomical; infection in these cases is intelligible. It is the *likeness* that suggests infection, is direct evidence for it and gives meaning to it. In the case of tuberculosis this anatomical evidence is signally wanting; it is the *unlikeness* between tubercle and all the things (except tubercle itself), including caseous matter, that are supposed to produce tuberculosis in experimental inoculations and that are inferred to produce it in man which has to be accounted for. Now, on an older view caseous matter was tubercle, and no other source than matter which had been tubercle was allowed. The difficulty is only evaded when the doctrine assumes, as it has assumed, this form: that tubercle is an inflammatory growth, the anatomical result of an inflammatory process; that this process is set up by the particles of some inflammatory product, itself the anatomical result of a

foregoing and primary inflammatory process. The conclusions here are founded upon analogy. Yet on these grounds of analogy it has been asserted as in the highest degree probable that *any* inflammatory product may, under certain circumstances, constitute a focus of infection and give rise to a tubercular process. If the analogy is a good analogy, which is open to question, no conclusion from analogy can amount to the highest degree of probability. Argument from analogy might very easily degenerate into hypothesis whose conclusions are not in agreement with the real facts. We cannot assume likeness between two things in order to conclude from the likeness that the things bear a causal relationship. Now, it seems to me that if we go back to the older views for an explanation and definition of infection, we should fall into error; we should have to assume likeness where it is certainly very hard to believe that any likeness can exist. A predisposition on the part of the tubercular tissues seems to me to be quite as intelligible and lawful as is an infective quality on the part of inflammatory or other products. In the one case it is an impress and tendency on the part of the tissues; in the other case an impress and tendency on the part of the agent. What is this impress and tendency on the part of the tissues produced by infective agency—call it impregnation or by any other name we choose—but a predisposition to a certain form of growth? If we say that all the things which become recipients of infective qualities and which influence tissues shew the tendency to undergo the changes proper to caseation; that the tubercular growth reflects its ancestry, or would do so if time permitted, by going through similar changes to those which characterise its prototypes; this may be taken to mean that the scrofulous tendency and



state is a first condition of the whole subsequent series of events—inflammatory and other processes, infection and tubercular growth.

We come now to the question whether fever may be in consequential relations with scrofula. May fever have an operative concern with the tuberculosis through that which the caseous matter represents—the underlying strumous state—and thus any assumption as to an infecting agent be avoided? We know that the tissues of our boys and their *modus vivendi* were universally stamped with special attributes and tendencies. Fever may, it is conceivable, be the means or agency by which an active development of these attributes and tendencies takes place in the special direction, tuberculosis, rather than in that other direction, caseation of inflammatory products, which under different or modified circumstances would be followed. We have still the facts, based on repeated observations, that tubercles and cheesy collections are found very generally together in the same bodies, and that both of these are very generally associated with the strumous or scrofulous state. Can we pass backwards beyond caseous matter, even beyond the processes by which caseous matter was brought into being, and connect fever with the natural tendencies of the strumous state, of which caseation and its result is but the mark and expression? If so, then we can place scrofula and caseous matter and tubercles in a series whose sequence and connection is made intelligible and consequential by the interposition of fever.

And first, let me relate a very interesting case which bears upon the question in an especial manner. Although it might be described as a case which is adapted to fit everybody's view, yet if taken in conjunction with the

other four cases it may throw light upon the relations between struma and caseous matter and tubercles, and the concern of fever with these.

A boy, aged 13, from the same Industrial School, came under my care at the Infirmary in September, 1876. He had been ill for a fortnight, and had been fevered from the first. I observed him only for three days. During that time he was insensible, and his symptoms were almost wholly cerebral and not remarkable. The following appearances were disclosed at the autopsy:—

The pia mater of the convexity injected; between the layers of the arachnoid, at base of cerebellum, a greenish-yellow lymph; arachnoid at posterior perforated space thickened and covered with the same sort of greenish-yellow lymph, the thickening and lymph extending backwards to pons varolii; base of velum interpositum also thickened and injected, and between its layers the greenish-yellow lymph; ventricles greatly distended by limpid serum; choroid plexuses much injected; brain otherwise healthy; no sign of tubercle anywhere on close inspection with an ordinary lens. Microscopic examination shewed marked cellular infiltration (small granular cells) of the pia mater where there was thickening and lymph; dense nucleation of its fibres; here and there, on the walls of the arterioles, little groups of granular cells and free nuclei, deeply stained by the logwood used in the preparation of the specimens.

In the lungs—several communicating cavities, having thick fibroid walls, in the apex of the right lung; marked peribronchitis fibrosa in the vicinity of the cavities; no pleural adhesions; no tubercles in lungs or pleuræ. On each side of the thorax, moulded between the compressed lungs and the diaphragm (in the pleural cavities), a large collection of greyish-white cheesy matter, easily scraped off from the pleural surfaces; these two collections of cheesy matter united, behind the œsophagus, by an isthmus of the same kind of material; no breach of continuity in the pleuræ.

In the abdomen—general, but not firm, matting of all organs by cheesy material; liver firmly adherent to the diaphragm throughout; a cheesy mass, four inches in diameter and half an inch thick, immediately under the central tendon of the diaphragm; in the right flank two cheesy masses, the size of large peas, adherent to the peritoneum



by a thin melanotic membrane and surrounded by a zone of peritoneal injection; a similar nodule, adherent to the mesentery; mesenteric glands enlarged and indurated, none caseous; small encysted collections of caseous matter in the liver and kidneys; abdominal organs otherwise healthy; no tubercle within the abdomen.

Such cases as this last are usually regarded as cases of scrofulosis and not tuberculosis. I have seen in the brain the ordinary products of a scrofulous meningitis in a comparatively firm caseous state; yet the clinical phenomena closely resembled those of tubercular meningitis, and no tubercles could be discovered anywhere in the body. But, the microscopic appearances observed in the pia mater of this fifth boy have been regarded as indicating the beginnings of tubercle.\* The marked cellular infiltration of the perivascular tissue and the nuclear development and aggregation along the vessels are said to be the essential factors in the earlier stages of a process which goes on to the formation of visible miliary nodules; and these changes are mixed up with the changes proper to ordinary inflammation. Further, the origin of the nuclei is affirmed to be partly in the connective tissue supporting the endothelium and partly in the endothelium itself.

Rindfleisch says that "the time is past when one can be satisfied with proclaiming 'miliary' tubercles as the specific product of tuberculosis;" and that "the definition of tubercles as a new-growth made up of small cells is entirely insufficient." He describes a lymphadenoid tubercle, a distinct variety of tubercle, occurring, as a rule, in scrofulous persons; he terms this form, "a peculiar large-celled germinal tissue." But, he says:—"I consider this tissue to be the acme of the process, *which*

\* Mr. Greig Smith, who kindly prepared and examined the specimens, so regards them. See his paper on *Acute Miliary Tuberculosis*, in the *Transactions of the Bristol Medico-Chirurgical Society*, Vol. I., p. 56.

*is not always reached.* This large-celled tissue usually forms the middle portion of the sub-miliary nodules, while at the periphery there prevails a small-celled inflammatory growth which gradually becomes continuous with the normal connective tissue." He then defines the tubercle of scrofulous persons as "a small circumscribed focus of scrofulous inflammation;" and he adds, "perhaps produced by a minute irritating substance introduced into the tissues." He speaks of "the histological identity of the scrofulous and tubercular new growths," and of the difficulty, in regard to a given tubercular lesion, in determining how much is inflammatory and how much is tubercular. Again, Rindfleisch says, "it is from the fixed cells of the vascular connective tissue system that the miliary tubercles, in my opinion, originate;" and, "when we find that the specific products of scrofula are developed with the aid of the permanent cells of the organism, it strengthens our belief that scrofula is a disease of the entire vegetation." Finally, he expresses the opinion that "miliary tubercles, apart from their etiology, are only small inflammatory foci, and in scrofulous persons only minute foci of scrofulous inflammation."\*

Now, putting the facts of this fifth case and the interpretation of the microscopic appearances alongside of the views expressed by Rindfleisch,—it might well be asked whether the general features of the case and the changes in the pia mater would not perfectly accord with the view that Rindfleisch takes? Is there anything in the lesions of the pia mater that could not belong to scrofulous inflammatory infiltration and scrofulous new-growth—call the latter tubercle or by any other name we please? But I would go further, and ask, were these lesions

\* *Ziemssen's Cyclopædia of Medicine*, Vol. V., p. 642, et seq.



special in any other sense than as being part of a scrofulous inflammation set up by an irritant like fever? Were these lesions anything but a part of a scrofulous inflammation already existing in the brain? Apart from the infective and re-sorption theory, there seems no reason for supposing that the general meningeal inflammation was due to the anatomical changes going on in the pri-vascular tissue and in the walls of the arterioles.

May we not, by help of fever and this last case, discard the view that the tubercles of these boys were a form of morbid growth with a separate and special histological character requiring the assumption of something so inclusive as "infective" to account for the circumstances under which they originated? At least we seem to have before us, irrespective of infection, anatomical developmental changes which are akin to inflammatory or embryonic growth, which are dependent on irritative circumstances and take place in scrofulous subjects in whom scrofulous processes had already resulted in caseous degenerations. I think the foregoing considerations give us definite suggestions as to the explanation of the facts of all our cases and their consequential relations; at least they point out a line for further enquiry and observation. I will briefly indicate this explanation and this line.

The strumous constitution is a real thing to us. Clinically we can realise it, and by what it is to us we can trust ourselves to recognise it in its several varieties when it comes before us in living examples. Pathologically, the characteristic of the strumous constitution is found in the characters of tissue-life and of cell-life in particular. Tissues are vulnerable and easily incited to morbid action, but to peculiar modes of morbid action.

The characters of cell-life are those of a low type of vitality, an incapacity for that reactionary metabolism and development, or for any of those things which we associate with healthy reaction against untoward influences and disease. The character of the development in scrofulous inflammatory exudations is of an especially low order; there is no reactionary development in the proper sense of the term. Instead, we note only a capacity for hyperplastic growth—numerical repetition—arrest of this low grade of development at a certain point, and then speedy death and degeneration; we have inordinate cell-production and thence caseation of the products. The process and progress of disease is slow in the strumous, and inflammation is oftener chronic than acute. Now, we may say that in most chronic strumous inflammations there is no fever, and that caseation is the rule. Nevertheless, in the strumous we may find fever of moderate degree with suppuration (erethitic scrofula); but then, a tedious course, intractability, recurrence and alternations of pseudo-acute with chronic conditions leave no doubt about the right choice of the terms “chronic” and “apyrexial” to denote the course and character of inflammations in the strumous. The strumous constitution is obnoxious to fever. Yet fever in higher degree may affect the strumous body, and in its various modes of secondary or sympathetic or inflammatory fever, of hectic and of specific fever, whether rheumatic or exanthematous, or any other specific form.

May we not, by the help of this fever, explain the occurrence of tuberculosis in the strumous? I can conceive that caseation and tuberculisation are different modes of the same tendency—the strumous tendency. The absence or presence of fever, the degree of fever, the



kind of fever and its intrinsic concomitant circumstances determine the mode and the result.

Chronic inflammation pursuing its course without fever goes on to caseation of the products. Even a moderate degree of fever may be allowed without affecting the result, for the reactive response comes slowly and with difficulty in the strumous. But let the fever assume a severer type and attain a higher grade, or let it be protracted only, being moderate in degree. We may fairly conclude that the products of febrile changes would be, in the strumous, if not more abundant, yet produced and circulating under conditions which would make them more irritative and harmful than in the non-strumous; these products can hardly be denied a greater opportunity for producing irritative effects in the strumous state than they have in the non-strumous state.

In this view, then, fever opposes activity to the inactivity of the scrofulous state; irritative reaction to strumous incapacity. Fever awakens what vitality may be possible in cells and tissues; it provokes as much reaction as these are able to put forth. Is tubercle, or what we may call tubercle, the result of all this? An attempt—a strumous and therefore imperfect attempt—at a reaction, akin to an inflammatory reaction, against the irritation of fever? The histological characters and anatomical circumstances of the scrofulous tubercle would very well accord with this view. Tubercle would then become a kind of embryonic tissue, the anatomical result of a normal reaction (so far as an imperfect and partial reaction could be called normal) to inflammatory irritation set up by fever in the strumous. The fact that tubercle has been found in caseous lymphatic glands (Schüppel) tends to support the view. Inflammation without fever or with

moderate degree of fever is enough for caseation ; and whilst in some persons a slight inflammation is accompanied by considerable secondary or sympathetic fever, in the strumous it is otherwise. In the cases where tubercle has been found along with caseation in the same parts, there has been enough of fever to produce the granulation or embryonic tissue which is the attempt at reaction on the part of the tissues. Again, it is said that tubercles are always found at the bottom of those scrofulous ulcers which are formed in glands by liquefaction of cheesy matter and inflammation in the vicinity ; that on the appearance of sound granulations the tubercles disappear and the ulcer heals (Birch-Hirschfeld). Here, also, it would seem as if the tubercles were the product of an impotent attempt to do what under subsequently occurring and more healthy conditions may be and is sometimes completed in the usual manner.

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CASE I.	CASE II.	CASE III.	CASE IV.
A. D—, æt 13. Duration about 24 days; typhoid state.	H. H—, æt 14. Duration about 30 days; delirium late; early copious diarrhæa.	W. A—, æt 12. Duration about 24 days; ptosis of right eye; left-sided convulsions once.	H. B—, æt 14. Duration about 17 days; lengthened coma; ptosis of right eye; dilatation of right pupil.
Arachnoid and pia mater thickened and opaque in S. fissure and generally; lymph in fissure.	Thickening and opacity of membranes in S. fissure and base; lymph on surface of left parietal convolution and in fissure. Tubercles in fissure; at base.	Thickening and opacity of membranes in S. fissure and base backwards to medulla; lymph in fissure. Tubercles in fissure; at base.	Thickening and opacity of membranes in S. fissure and slightly at base; adherent to dura in places; lymph in fissure. Tubercles in fissure; at base; a few in choroid plexuses.
Patch of tubercles in fissure; in course of vessels generally; in choroid plexuses.	Slight excess of liquid in ventricles.	Ventricles dilated; excess of liquid.	Little liquid in ventricles.
Ventricles dilated; excess of liquid.	Both lungs, tubercles throughout.	Right lung, tubercles throughout; caseous mass at apex.	Both lungs, tubercles (not numerous) throughout.
Left lung, tubercles throughout.	No tubercles in pleura.	Right pleura, recent adhesions; tubercles over whole pleura.	Both pleuræ, tubercles all over.
Left pleura, on diaphragmatic layer, a patch of tubercles.	Bronchial glands on both sides caseous.	Right bronchial glands caseous.	A left bronchial gland caseous.
A left bronchial gland caseous.	Ulcerated Peyer's glands in ileum generally and at valve; hyperæmia (slight) of large intestine; tubercles in duodenum.	Hyperæmia of ileum and cæcum; ulceration of Peyer's glands at valve and for two feet upwards; no tubercle.	Ulceration of Peyer's glands in ileum generally and at valve; ulceration of large intestine at intervals; no tubercle.
General hyperæmia in patches in whole intestinal tract; ulcerated Peyer's glands at lower end of ileum and at valve; no tubercle.	Liver, tubercles on surface.		Peritoneum of diaphragm, liver, spleen, intestine and abdominal wall studded with tubercles and melanotic in places.
Liver congested; tubercles on surface.	Spleen, much infiltrated with tubercles.		Liver congested; tubercles in substance.
Spleen, a patch of tubercles on surface.	Both kidneys infiltrated with tubercles.		Spleen, slightly infiltrated with tubercles.
Right kidney, a few tubercles on surface (under capsule).			Both kidneys, a few tubercles in substance.

## CASE OF MR. CROSBY LEONARD.

BY DRs. FOX AND SHINGLETON SMITH.

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IN 1874, whilst driving in his mail phæton, Mr. Leonard turned his head to an unusual degree to look at something behind him. Whilst he was in this position his horses started away rapidly, and he immediately felt a jar or wrench in the middle portion of the cervical region. From that moment until his death in October, 1879, he was never free from discomfort. This was manifested at first with the symptoms of an ordinary stiff neck; he was unable to turn the head toward either shoulder without considerable pain, and the act of sneezing intensified this pain to an almost excruciating degree. The first symptoms, therefore, persisting alone for nearly twelve months, were muscular spasm of the cervical muscles, and pain, the latter augmented by any movement of the cervical spine. After this period the stiffness of the neck gradually subsided, and the local pain became much more moderate. The power of holding the head erect had remained normal. In the summer of 1875 he fell down in his own garden, and fractured the left tibia and fibula.

As he began to move about with a crutch he complained of numbness of the left shoulder, arm, hand and

fingers, with occasional tingling. At first he fancied that these symptoms were caused by the pressure of the crutch in the axilla. Slightly subsequent in point of time to these sensory phenomena he experienced a certain difficulty in raising the left arm, as in the action of putting on his coat, and also some awkwardness in minute actions with the fingers of the left hand, such as buttoning his clothes, the latter due partly to the impaired sensation in these organs. During his convalescence, also, he was concerned to find that he could not use the leg as freely as should have been the case in a limb that had recovered from fracture; and this fact, with certain sensory phenomena in the leg (formication, numbness, imperfect sense of the hardness of the floor, &c.), shewed that this limb was gradually sharing in the paretic condition of the left arm. He was able to move every group of muscles in the leg and to walk tolerably well, but stairs were becoming a difficulty to him, and there was a faint dragging of this left leg, unless he made an effort. Matters remained pretty much in this condition for a year, during the whole of which period Mr. Leonard performed his duties as Surgeon to the Infirmary. In the early part of 1877 he found a slight diminution of motor power and of sensation in the right arm and hand. The sensory phenomena increased in intensity as the year went on, and in October, 1877, he felt Charcot's points as one at a distance of an inch and a half, both vertically and transversely, in the right hand. In this respect the left hand was no better and no worse than the right. He had been able, however, in January of that year to perform several operations; at the end of March he had reduced an obturator dislocation of the femur by manipulation; in the middle of April he



operated on *nœvus* by ligature; and as late as September 18th performed his last operation—excision of the eyeball.

In October, 1877, the note of his condition was as follows:—General health perfect, except that the bowels are constipated and that he gets occasional severe headache, to which he has been subject for many years. Nerves of special sense normal, except that the sense of touch is impaired (not lost) in both arms and hands and in the left leg and foot.

Motor power is deficient in the left arm and hand; he cannot button with the left hand, and he grasps very imperfectly. In the left leg he uses all the muscles of the leg, if he attends to them, but in walking he scrapes the ground, if he is not thinking of what he is doing. There is slight loss of power in the right hand and arm; but he can button and grasp with it, and writes very well. The right leg not affected.

No paresis of bladder or rectum.

A little wasting of the muscles of the left thumb and of the left deltoid.

No tenderness of any spinous process or along the heads of the ribs. No projection of spine.

No tremor; no contracture; some cramp occasionally in the left leg, and the pain accompanying the cramp was felt acutely.

No increase of reflex excitability.

No evidence of vasomotor disturbance, except a tendency to redness of face and neck (which was not unusual to him on slight exertion or excitement at any time), and just the commencement of *œdema* of the ankles of both legs.

The Faradic current was hardly felt in the hands and

arms, and induced very slight muscular movements. The continuous voltaic current, on the other hand, seemed to cause so much pain, that it had to be abandoned as a means of treatment.

In the spring of 1878 the right leg began to shew the same phenomena as the other limbs. There was some numbness in it, some difficulty in raising the leg, except with the full attention of the patient.

During the winter 1877-78 Mr. Leonard had become very stout, mainly owing to the increasing difficulty in locomotion; but his general health was good, and his mental faculties bright and unclouded.

In April, 1878, he went to the brine baths of Droitwich, and he always thought that he had been weakened by them. It became difficult for him to get into a low carriage, and he always felt jarred and more numb after driving out. Stairs, too, became less and less easy.

As the autumn and winter of 1878 came on he was increasingly sensitive to cold. The left arm became almost inert; he was unable to raise a fork with the left hand; and the left leg became decidedly less useful. The anæsthesia of the right hand increased, and he was unable to feel a pen between his fingers. It was, however, a remarkable point (interesting as bearing on the connection of sensation with co-ordination of motion), that to within a few days of his death he was able to write very legibly, if only he kept his eyes on the fingers that held the pen. It is possible that some of the difficulty in moving the legs was due to their weight, both from the increase of fat in them and from the œdema, which during the latter months of his life was considerable.

This increased œdema and occasional palpitation of

the heart were the only signs of vasomotor disturbance. The pupils were always regular and normal and the sight good.

For some months before the end he remained upstairs, to avoid the difficulty of ascent and descent; and to the last day or two of his life he was able to walk, with help, from one room to another on the same floor.

Strange to say, in September, 1879, he had to some extent regained the power of raising the left arm; and on the day before his death he walked into his bedroom with his sister's help, as he said himself, "like a soldier."

He never suffered from decubitus, cystitis, affections of the sphincters, or any cerebral symptoms.

The account of the last few days of his life are given below by Dr. Shingleton Smith.

It is needless to say that Mr. Leonard tried with a patient perseverance, which was only a part of his noble character, a great variety of remedies. Local counter-irritation in various forms, Mercury, Biniodide of Mercury, Iodide of Potassium, Tonics, Strychnia, Ergot, the continuous current, the Droitwich baths, and many other remedies were tried without benefit. The nature of the lesion and the probable course of the disease rendered all advice as to therapeutics half-hearted; and his own pathological knowledge deprived him for years past of any real hope of permanent recovery.

The symptoms in their course seemed to make the seat of the lesion, and to a great extent its nature, fairly clear.

1. Injury, probably at the commencement a minute hæmorrhage, high up in the cervical spinal membranes.

2. Irritative meningitis at this spot, very localised, evidenced by pain on movement of the cervical muscles.



3. Symptoms of very gradual myelitis from compression, always incomplete.

4. Finally, ascending degeneration reaching up to the medulla oblongata.

Apart from the rarity of the primary lesion, and the still more rare localisation in such narrow bounds, it seems remarkable that the diaphragm was not affected, although the lesion was so close to the origin of the phrenic.

E. L. F.

On October 11th, 1879, a bottle containing several ounces of urine, was sent to me by Mr. Leonard, with a request that I would examine it for him. Its condition was as follows:—

Sp. gr. 1037, high coloured but clear, not smoky, no sediment, no reaction for bile with nitric acid, slight cloudiness on boiling which did not disappear with acid, and slight sediment subsequently collecting, with liquor potassæ on boiling a deep brown discolouration, and with solution of copper and liq. potassæ the red precipitate characteristic of the presence of sugar. On filling the saccharometer tube for polarisation the fluid was found to be too deeply coloured to allow light to pass, and after filtration through animal charcoal three times it was still too opaque for the quantity of sugar present to be estimated.

On Oct. 13th, sp. gr. was 1034, colour deep, a trace of albumen present and much sugar.

Oct. 11th. The condition on this day was as follows:—Sitting up as usual in the ante-room adjoining bedroom, intelligent as usual, but rather apprehensive that his condition was much worse. He complained that two days

before, *i.e.*, Thursday, October 9th, he lost his appetite, and had been unable to take solid food since; he also felt an intolerable thirst with feeling of dryness of throat and lips; the thirst was very trying, gave him great discomfort, and nothing seemed to allay it. He also complained of feeling low, and thought the heart's action was weak. On auscultation the heart sounds were normal, and air entered the lungs freely down to the bases on taking a deep inspiration, which he seemed able to do fairly well. The radial pulse was small and weak, about 108; no irregularity or intermission was observed. The face looked more flushed than usual and the skin of the neck was much injected, but there was no evidence of deficient aeration. Both legs were considerably œdematous, and the wrists were slightly so. He complained of great fulness and tightness across upper part of abdomen. There was visible abdominal enlargement from flatulent distension and partly from the presence of abundant fat. Bowels had not acted for three days, and for months had not done so without a powerful purge. It had been noticed as a curious fact that there was more power in the left arm than before: he was able to raise the arm up to the level of the head without help, but the right arm was a little worse; there was still considerable power of movement in the right arm, he could write with a pencil in a legible manner and seemed to use the hand fairly well for most purposes, but could not grasp powerfully, and had a difficulty in holding a cup or glass for drinking. Neither leg was completely paralysed; both could be raised off the ground whilst sitting, but they were not able to give support in standing. No spasmodic twitchings were observed; reflex excitability was not marked.

There was not, and had not been, any difficulty in

holding or in passing the urine, and the bowels had never acted involuntarily; the quantity of urine passed was not large, about a pint and half twice in the twenty-four hours, night and morning, none was passed except at those times.

On being told that the urine contained sugar, he remembered that about a month previously some white spots on the slippers were seen which could not be explained, but which no doubt arose from the evaporation of a few drops of saccharine urine sprinkled upon them; this was the only guide as to the duration of the diabetic symptom; the thirst had not existed till two days ago when the appetite failed.

The flushing of the face and neck had been observed for a long period and had become persistent. No difference or irregularity of pupils had been observed, and no complaint of any defect of vision was made.

On the following day, October 12th, he was sitting in the bedroom, not feeling well enough to be moved to the adjoining room, the bowels had acted freely in consequence of a purgative dose given the previous night, and there was less feeling of distension of abdomen. Thirst continued as on day before, there was no appetite, and he felt so low that he was obliged to take brandy repeatedly.

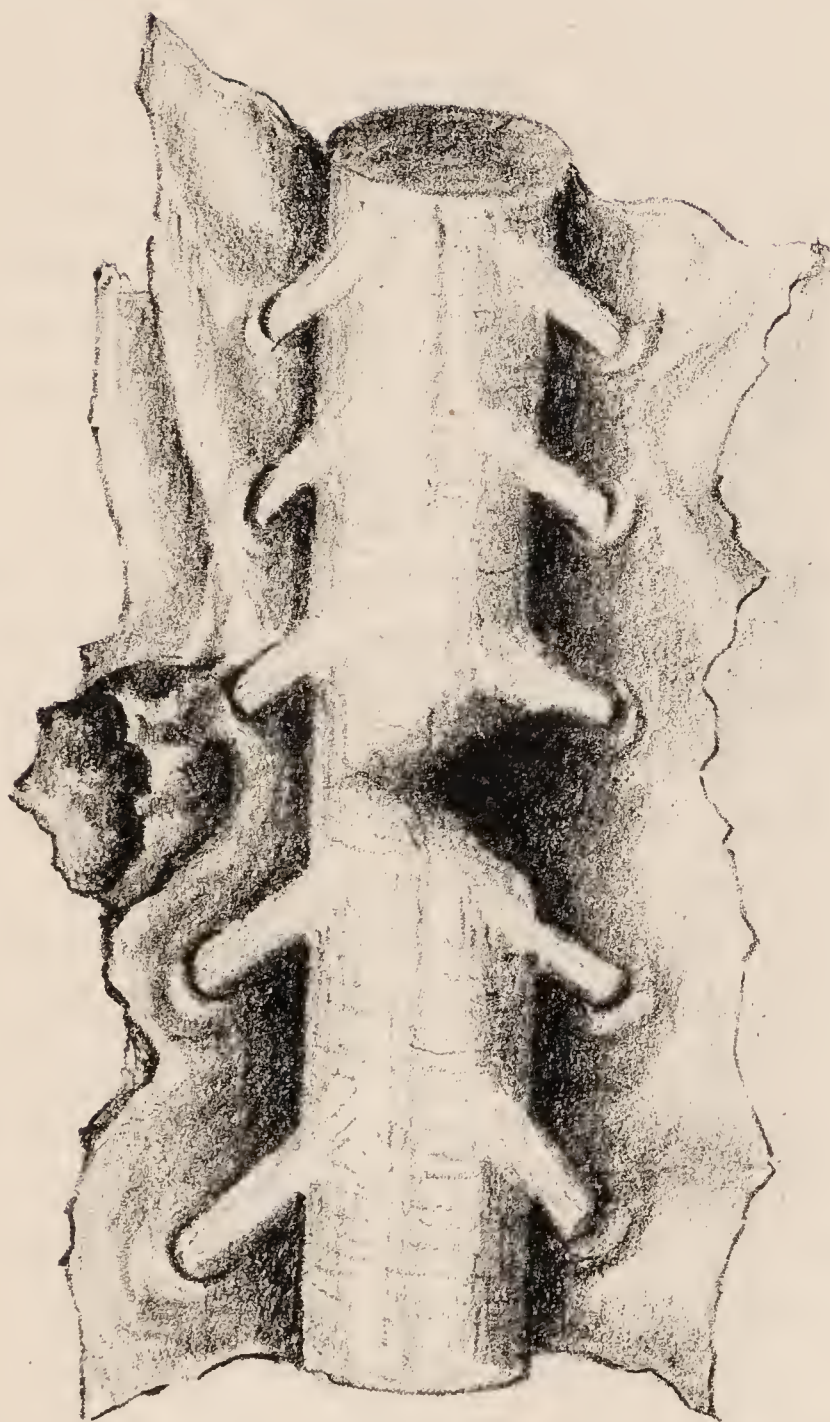
October 13th. Had slept fairly through the night, but always longed for the morning, was got up as usual at eight o'clock and was helped to the chair in the middle of bedroom, but did not feel able to go into ante-room. He felt weaker and took stimulant repeatedly, but did not feel able to eat anything. He was uneasy as to the condition of heart, but on examination nothing abnormal was detected. The pulse was small and weak, about 120, scarcely perceptible at either wrist in consequence partly



of œdema. Breathing seemed an effort, but respiration was not much accelerated; a frequent deep breath of a sighing character was noticed. He was able to converse, but said that he felt too unwell to take any interest in the newspaper, which he had hitherto read with pleasure. Mentally, and as regards the limbs, there was no change, and at the time of being visited he had been sitting in the chair with head unsupported for eight hours, and preferred this position to the bed. He had felt more helpless than ever before, and was uneasy as to the difficulty in getting back to the bed at night. This difficulty was surmounted at about half-past nine, but he felt thoroughly exhausted, and said he was faint but should be better presently. After this he was helped on to the night-stool, the bowels acted, his head fell back, he lost consciousness, and on the arrival of his medical attendant a few minutes afterwards no signs of life remained.

*Post-mortem examination*, October 14th, 1879, 18 hours after death. The spinal canal was the only part examined. Body externally presented no appearance of bed sore, not even redness of the skin from constant sitting; there was considerable œdema of both legs and slight swelling of both upper extremities. Abdomen was very large from adipose tissue chiefly, partly also from flatulent distension of intestines. The subcutaneous fat was very abundant, at the back of the neck being nearly two inches in thickness. Skin of neck and face generally was flushed from capillary dilatation.

After opening spinal canal in its whole length nothing abnormal was visible from the posterior aspect; the dura mater had its normal appearance and no growth appeared to exist between it and the bony walls. On touching the cord with the finger it appeared to be softer at the middle







of the cervical region than elsewhere; everywhere below it had its normal consistence. On cutting through its attachments and raising the cord enveloped in its sheath, commencing from below, nothing abnormal was noticed below the middle of the neck, but here a mass of reddish jelly-like substance came into view, situated on the anterior aspect of the cord outside the dura mater, between it and the bone; this mass of soft pulp was taken away and the whole cord was removed, being cut through opposite the first cervical vertebra. On examining the anterior aspect of the internal surface of the dura mater it was found that this membrane was thickened and its surface roughened; small masses of reddish jelly-like substance were visible imbedded in a reticular connective tissue, these small masses being in appearance exactly like that which had escaped in the removal of the cord. The thickening of dura mater extended through a length of about an inch vertically; the jelly-like substance was collected, for the most part, into one mass, but other small portions were distributed around. On cutting through the dura mater and exposing the cord itself, a decided indentation in its left anterior surface was visible, caused by the pressure of the mass on the surface of the sheath; at this spot the cord was much constricted and probably not more than half its usual diameter; the indentation was entirely on the left side and was not visible posteriorly. No abnormality of the inner membranes was present and no morbid growth was seen inside the dura mater sheath. The sections of cord itself shewed little naked-eye disease; the grey matter was pale everywhere, and just above the constriction the whole area of section was softened, but it was nowhere diffuent.

The cord with its membranes was immersed for some

weeks in Müller's fluid. The following is a description of the specimen after being thus preserved:—The dura mater much thickened and roughened in its left anterior aspect for two inches in length, commencing immediately above the lower cervical enlargement. One large cyst sac still visible at the centre of the length of thickened dura mater, the cavity of the sac being as large as a hazel nut, its lining membrane smooth, its inner wall (that presenting towards the cord) thinner than the outer and much wrinkled in consequence of collapse of the sac following escape of its contents. On reflecting the dura mater the inner wall of the cyst visible as a circular wrinkled patch raised above the surrounding smooth arachnoidal aspect of the dura mater. The outer surface of the dura mater presented other smaller cyst-like structures, but less defined and looking like mere meshes in the connective tissue, with no definite cyst walls, containing a small quantity of reddish myxomatous tissue resembling that which had been removed from the large cyst.

The cord itself presented a deep indentation on its left anterior surface, evidently caused by the pressure of the cyst in the dura mater. The middle line of the cord was displaced a little to the right, but the right half of the anterior and lateral columns presented no indentation or visible atrophy. The part compressed was much softened; after two months immersion in Müller's fluid half-an-inch at the site of compression continued to be soft to the touch, an inch above and an inch below hardening was perfect. In section at the centre of the indentation no normal structure was visible; the mass presented a semi-diffuent aspect with no difference between white and grey matter. A quarter of inch higher the whole area of the section had a gelatinous

aspect, as if infiltrated with serum; the horns of grey matter were perfectly distinct, but the central grey mass on the left was evidently softer than on the right. The left lateral column appeared healthy, but the deeper part of the posterior columns in contact with the left posterior horn was paler than elsewhere.

Higher up no abnormality was visible other than a general gelatinous appearance; no defect was manifest either in the lateral columns or the columns of Goll.

Below the seat of the pressure more degeneration was visible than above it. Three quarters of an inch below there was a patch of much infiltrated tissue occupying the posterior column on the left side (the posterior root zones), but limited by the posterior median fissure, extending along the posterior horn to the surface of the cord; no degeneration of the lateral columns was seen. An inch below the whole area of section presented the normal appearances.

Sections of the cord prepared by freezing and subsequent staining in logwood exhibited the following features:—

In the upper cervical region, opposite the decussation of the pyramids, the normal distribution of grey and white matter (*Corps de Deiters*) was visible. No difference in the two halves existed; there were no patches of degeneration, but the whole tissue seemed to have a loose texture, the grey matter more particularly looked as if infiltrated with serous exudation. The epithelium of central canal had undergone abundant proliferation, the canal being blocked with a mass of small round nucleated cells, no columnar cells being present. The canal was surrounded by a number of concentric rings of connective tissue growth with very fine elongated nuclei. The



shape of the canal was peculiar, being much elongated antero-posteriorly, and tapering to a rather pointed extremity in front and behind; the connective tissue rings surrounding the canal did not pass continuously round, but were incorporated with the neuroglia tissue of the commissure. This part of the sections, viz., that surrounding the canal, was less stained than the tissue of the lateral or other columns. Scattered throughout the central part of the cord were numerous deeply stained amyloid bodies of large size. The grey matter had a porous, spongy texture, and with high powers many connective tissue cells, with tailed processes, could be made out (neuroglia cells).

In the sections through the cord, immediately below the cervico-brachial enlargement, the central canal was replaced by a mass of connective tissue infiltrated with small nuclei. The meshes of this tissue were coarse, and the cells were not very abundant. The mass was triangular in shape, the apex of the triangle being at the bottom of the anterior median fissure, and its long diameter was transverse. Immediately surrounding this central mass was a finer meshed connective tissue infiltrated with sharply defined amyloid bodies, and containing a few small elongated nuclei, but much less stained than the neuroglia elsewhere. The grey matter laterally had a very loose, spongy texture; neuroglia cells, with extremely delicate fibrillar processes, were scattered abundantly throughout, and the nerve cells appeared to be somewhat atrophied. The amyloid bodies were present in all parts of the cord, particularly around the margins and along the edges of the fissures, but were most abundant of all in the tissue surrounding the central canal.

In the middle dorsal and the lumbar regions similar

appearances were observed, viz., the proliferation in the central canal and the diffusion of amyloid corpuscles; but the grey matter presented a less spongy texture. The amyloids were most abundant in the lumbar region, where also they were larger and more equally diffused.

An examination of the mass of jelly-like substance, which escaped from the cyst in the dura mater when the cord was being removed from the spinal canal, disclosed the existence in it of cells numerous and various in form, mostly of an epithelial type and in a condition of myxomatous degeneration. The spheroidal form predominated, but many flattened, scale-like, and tailed, spindle and stellate cells were present. The size presented as great variety as the form of the cell, some being no larger than the ordinary kidney epithelium, whilst others were equal to the largest cancer cells. All the cells became readily stained by logwood and by carmine, and these colouring agents brought into view one or more small-sized nuclei in each of the cells; there was little variation in the size of the nucleus of the cells, which varied so much in size as a whole; the nuclei were a little larger than the colourless blood-cells; many groups and masses of small free nuclei were also seen; all the nuclei stained very deeply, although the surrounding cell-substance remained colourless; they were perfectly round, sharp-bordered, highly refractile, and some of them granular. The outer part of the cells was for the most part very transparent; in some specimens stained in carmine and mounted in glycerine jelly it became almost invisible, but in the logwood specimens it was more distinct. Many of the smaller cells consisted only of a tinted nucleus at the margin of a thin colourless capsule, containing quite colourless mucoid (?) contents. The larger

EXPLANATION OF PLATE XXII.

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- Fig. 1.—*Transverse section of the grey matter from lower cervical region of cord of C.L., shewing peculiar arrangement of the trabeculæ of fibrous tissue, the central canal occupied by proliferating cell elements and open-meshed fibrous network, and surrounded by concentric rings of fibrous tissue containing numerous amyloid bodies.  $\times 30$  diamrs.*
- Fig. 2.—*One of the large cells from the substance of the gelatinous mass embedded in the dura mater of cord, shewing a deeply stained network, connected with a central nucleus, traversing the clear cell contents.  $\times 1000$  diamrs.*
- Fig. 3.—*A connective tissue cell from the grey substance of the cord.  $\times 1000$  diamrs.*
- Fig. 4.—*Group of epithelioid cells from the same source as fig. 2.  $\times 200$  diamrs.*



Fig. 1

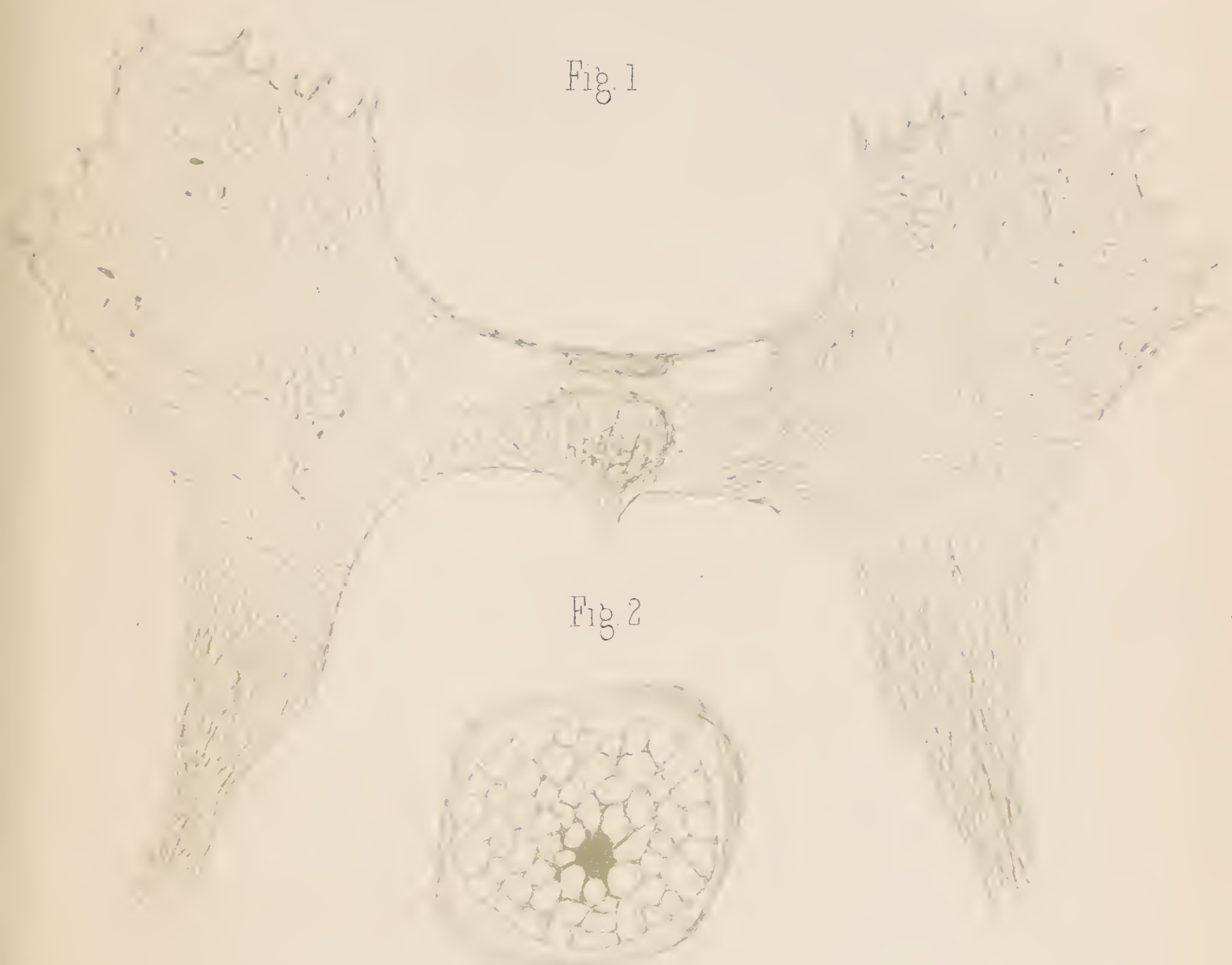


Fig. 2

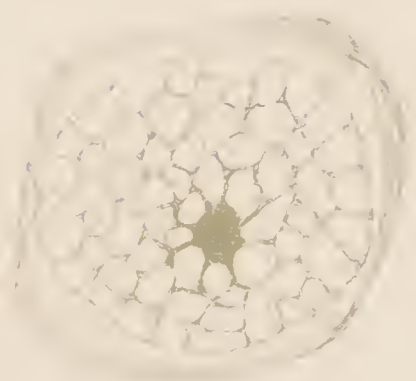


Fig. 3

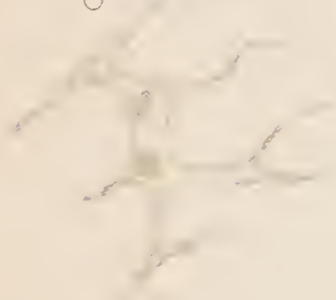


Fig. 4





cells presented a reticular stroma, dividing up the cell contents into minute masses with delicate partitions, the processes being seen to radiate from a central or in other cells a marginal nucleus. The structure of these cells exactly resembles that described by Dr. Klein as visible in most cells when examined by the highest objectives; in the specimen now described a power of 200 diameters was sufficient to demonstrate the network of fibrils disseminated throughout the colourless mucoid contents of the cell.

Little inter-cellular substance was present, and no fat globules or granular debris.

R. S. S.



# THROMBOSIS OF BOTH MIDDLE CEREBRAL ARTERIES.

BY DR. BRITTAN.

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THE following statement of the case and of the *post mortem* examination of the late R. W. Tibbits, whose premature illness and death in the autumn of '78 were matter of such deep interest to his colleagues and the members of the various professional societies, of which he was so prominent a working member, are now published in compliance with the wishes of many of his friends to whom the particulars of his case must be of special interest, as well as for the instruction they afford.

Mr. Tibbits at the time of his death had but attained his 36th year. As a student he was, in consequence of symptoms of phthisis pulmonalis, sent to the South of Europe. He returned in good health and vigour within the year to resume his studies. After passing his examinations, he accompanied a patient to the East, and whilst travelling in India suffered from some disorder of the liver. Soon after his return he became, and to his death continued to be, very actively employed in the public and private duties of the profession he followed with such energy and ardour, and enjoyed good health, with the exception of attacks of hepatic dyspepsia with scanty and high-coloured urine, which he considered to be the relics

of his Indian attack, due to want of and relieved by active exercise. He was a man above average height, strongly but rather slightly framed, capable of much exertion and of great endurance. He frequently would walk twelve to twenty miles after his day's work, which was always done on foot, and had only lately returned from a trip in the Swiss mountains, crossing over the Col du Geant from Connayeur to Chamounix in a severe storm, when seized with the attack that at once rendered him powerless. The only premonitory symptoms were, as he described them himself to me during his illness, that in the previous three months he was conscious of an occasional and very transient indistinctness of vision, and that when making clinical remarks to the pupils he had noticed a look of surprise, and on pulling himself together found he had been using a wrong and inappropriate word; on account of these symptoms it was, in fact, that he went for his Swiss holiday, from which time they disappeared.

On the night of Sept. 26, after a busy day, and being occupied some time in finishing a paper he was writing, his companion noticed an alteration in his speech, and, thinking he was over tired, induced him to retire to bed. In undressing he found he had lost the use of his left side; he was just able to complete his change with his right arm and throw himself on the bed, and call in his friend as he passed the door, and tell him he had had a "stroke." He was then troubled with frequent severe rigors and convulsive movements, but never for a moment lost consciousness. In this condition, paralysed in the left side and with altered speech, he remained for about three weeks, when power of movement began to return in the foot and leg, but with it severe pain in the anterior

crural nerve, in the poplitæal space, and between the great and adjoining toe. His appetite was poor, but he drank freely of his favourite beverage milk, or Swiss milk and soda water. His tongue was, as he termed it, a "cerebral tongue." His urine passed with some difficulty. He was free from pain in the head, and evinced remarkable composure and cheerfulness; from the first he noticed a peculiar movement and contraction of the fingers of the paralysed hand on gaping or yawning.

On Oct. 26 he complained of feeling very ill; his pains were severe, and his temperature ran up to 104, he had severe rigors and profuse sweats. Under the employment of quinine in full doses these symptoms abated, and he began to be able to move the hand and fingers slightly; but just as had been the case with the leg, so with returning power in the hand, severe pains came on about the thumb and shoulder joint. This pain continued, but in all other respects there was a great improvement, his tongue cleaned, his appetite returned, and he enjoyed solid food.

On Nov. 13 his temperature began to rise again to from 100 to  $101\frac{3}{5}$ , and he complained of feeling exhausted by this and the profuse sweating that accompanied it. On the 20th he suddenly became comatose, breathing stertorously, without any convulsive movement, but a strange thrilling sort of vibration through the right side, perceptible on grasping the bed post. In this state he remained till he died on the morning of the 22nd.

The endeavour during his life to account for such an attack in such a patient involved several considerations, and doubtless occupied his thoughts during his illness, though he spoke but little on the subject. One day, however, he asked me to talk it over with him. The



suppositions he suggested were—a morbid growth, an embolus, a thrombus, hæmorrhage? Without giving him any bias, as I thought, to my own view, I stated to him as they appeared to me, the *pros* and *cons* in each case, and was rather pleased that he adopted the view of hæmorrhage from some small ruptured vessel, as it offered, to my mind, more hope of restoration. Some days afterwards, however, he turned to me suddenly with the remark that he was confident I was of opinion that it was thrombosis, and he felt sure I was right.

My reasons for so thinking were as follows:—In the absence, as he assured me, of any hereditary or acquired constitutional taint, with no premonitory symptoms but the transient indistinctness of vision and the misuse of words, the want of any symptom of pressure, and the suddenness of the hemiplegia, I dismissed any morbid growth.

He had never suffered from rheumatism, nor any indications of vegetations about the cardiac valves, and therefore as between embolism and thrombosis I inclined to the latter. The question that remained then was thrombosis or hæmorrhage? The suddenness of the attack was like hæmorrhage, but then there was no unconsciousness even for a moment, and no symptom of pressure except on the one spot. It is true we see cases of obstruction with more or less unconsciousness, and of hæmorrhage when slight without unconsciousness; but I believe the statement made, and corroborated by high authority, that when “hemiplegia, complete and absolute, occurs suddenly without loss of consciousness it is due to softening and not to hæmorrhage,” to be generally true, in this case it certainly proved so; and, moreover, the premonitory symptoms just referred to, occurring some weeks before

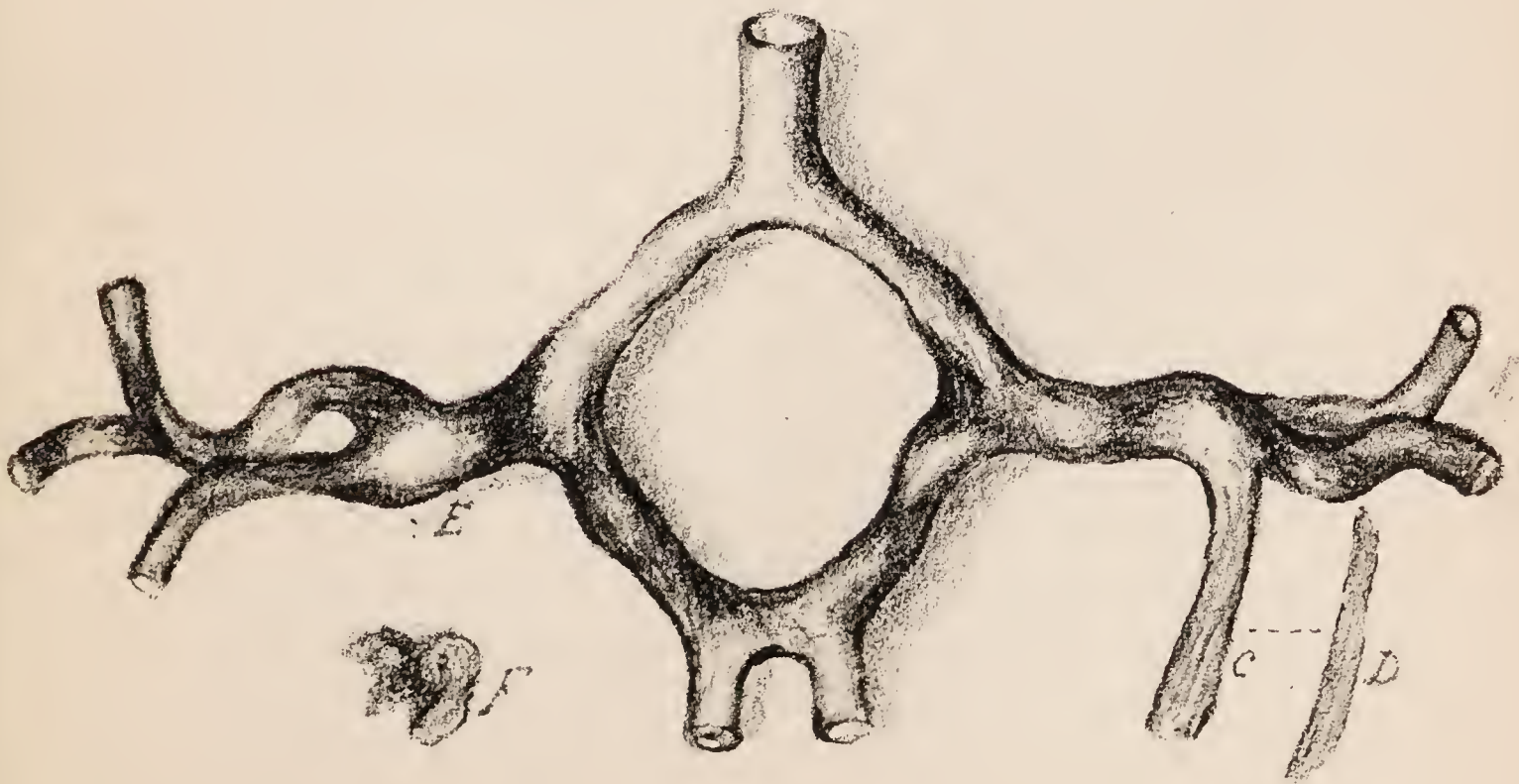
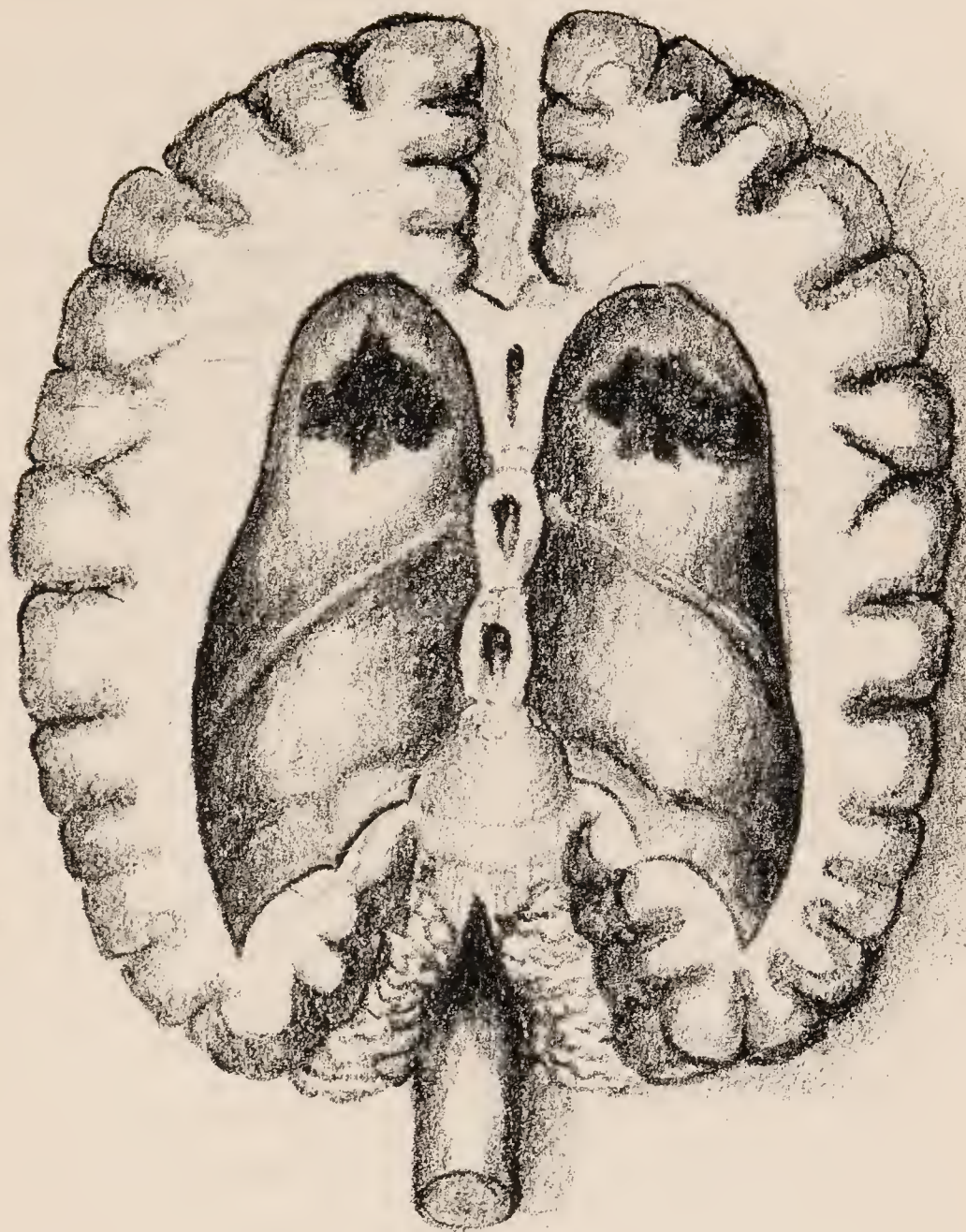
the actual seizure, were incompatible with the supposition of sudden hæmorrhage, whilst they exactly accord with that of thrombosis.

The attack of Sept. 26, then, would be the result of softening reaching a certain point in consequence of a block in the vessels supplying the portion of brain affected and on the right side. At what exact period this block became complete it is impossible to say, but certainly the premonitory symptoms affecting the sight and speech must be regarded as indications that even then the brain substance was undergoing a morbid change. It will be observed that on Oct. 26th he had a rise of temperature to 104, with rigors and profuse sweating; it was about this time that in all probability the block found in the left side also became established, and the damage in the brain substance on this side became important. Again, on Nov. 13 the temperature rose with exhaustion and sweating and rigors, the precursor of the complete abrogation of function on both sides, which culminated on the 20th in absolute coma, but with that peculiar thrilling motion or convulsion on the right side which now for the first time indicated that the left side of the brain was affected as well as the right.

The *post mortem* examination revealed the condition shewn in the accompanying drawing.

The circle of Willis, it will be observed, has been turned over so that A is the middle cerebral artery of the left, B of the right side. In the enlargement on A at E was a firm plug, shewn at F—in B at C the elongated mass D; both consisted of “fibrillating embryonic tissue.” In tracing the artery on the right side the brain substance broke down. On opening the ventricles, no extra fluid was found, but softening was obvious, extending as in the









parts shaded in the drawing—on the left side not so deeply, but on the right nearly through to the surface, where it broke down in tracing the artery.

It only remains with the *post mortem* results before us to decide whether the thrombus on either side proceeded from and was anteceded therefore by degeneration of brain and consequent affection of the remoter branches of the artery plugged, in which the coagulation therefore would be secondary, a view which seems to me under all the circumstances untenable. But or, on the other hand, if the thrombus was the primary element, what could have induced its or rather their occurrence symmetrically on either side? unless, indeed, the scanty and loaded urine and the hepatic dyspepsia, ascribed to a short residence in India, were really indications of gouty diathesis, and the thrombosis the result of gouty degeneration in the vessels.

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# ANEURISM OF THE ARCH OF THE AORTA OPENING INTO BOTH TRACHEA AND ŒSOPHAGUS.

BY DR. BRITTAN.

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The following case is remarkable for the very significant indications for diagnosis afforded during life—the grave lesions the aneurism caused—and the continuance of life under such perilous conditions.

The patient, aged 49, married but without children, was a tall well-made woman, who had been under my care for some years. There was an indistinct history of rheumatism, but her father and brothers and two sisters died of diseased heart; during the last 3 or 4 years of her life she suffered much from irregularity of circulation, with palpitation, and trying irritable cough; at times I have heard a slight and indistinct cardiac murmur, but generally there was no abnormal sound.

Having been for some weeks better than usual, on the 2nd of June she complained of a cough more paroxysmal in character than ordinary, and of spasmodic pains about the arms, chest and shoulders. In a week the cough became stridulous, moist sounds were audible in the trachea, but the lungs remained clear and she coughed up several masses of blood clot unmixed with mucus. Soon the spasmodic pains became more intense, and especially severe on attempting to swallow; still most careful ex-



amination failed to reveal any expected morbid sound, but at the back the heart sounds were very intense through the stridulous breathing and moist sounds which were loudest over the bifurcation of the trachea. There was also some tenderness in the spine, and when she swallowed, which she did with difficulty, a gurgling could be heard particularly at the same spot.

The symptoms now subsided for a fortnight. On June 30th I noted, pulse now intermits every 3rd or 4th beat, there is no abnormal sound audible in the front of the chest nor pulsation to be felt nor heard unusually behind. She coughs, but with very little expectoration, she can swallow better, taking fluids readily, solids only a bit at a time, otherwise the spasmodic attacks come on though less severely. Her breathing is much less stridulous, at times quite easy and clear. There is no œdema, but distension of the veins over the front of the chest, particularly on the left side. She complains of soreness in the fauces and throat.

On July 1st.—Any movement seems to induce severe spasmodic pain, her breathing is not so full, the left lung though perfectly resonant is not acting.

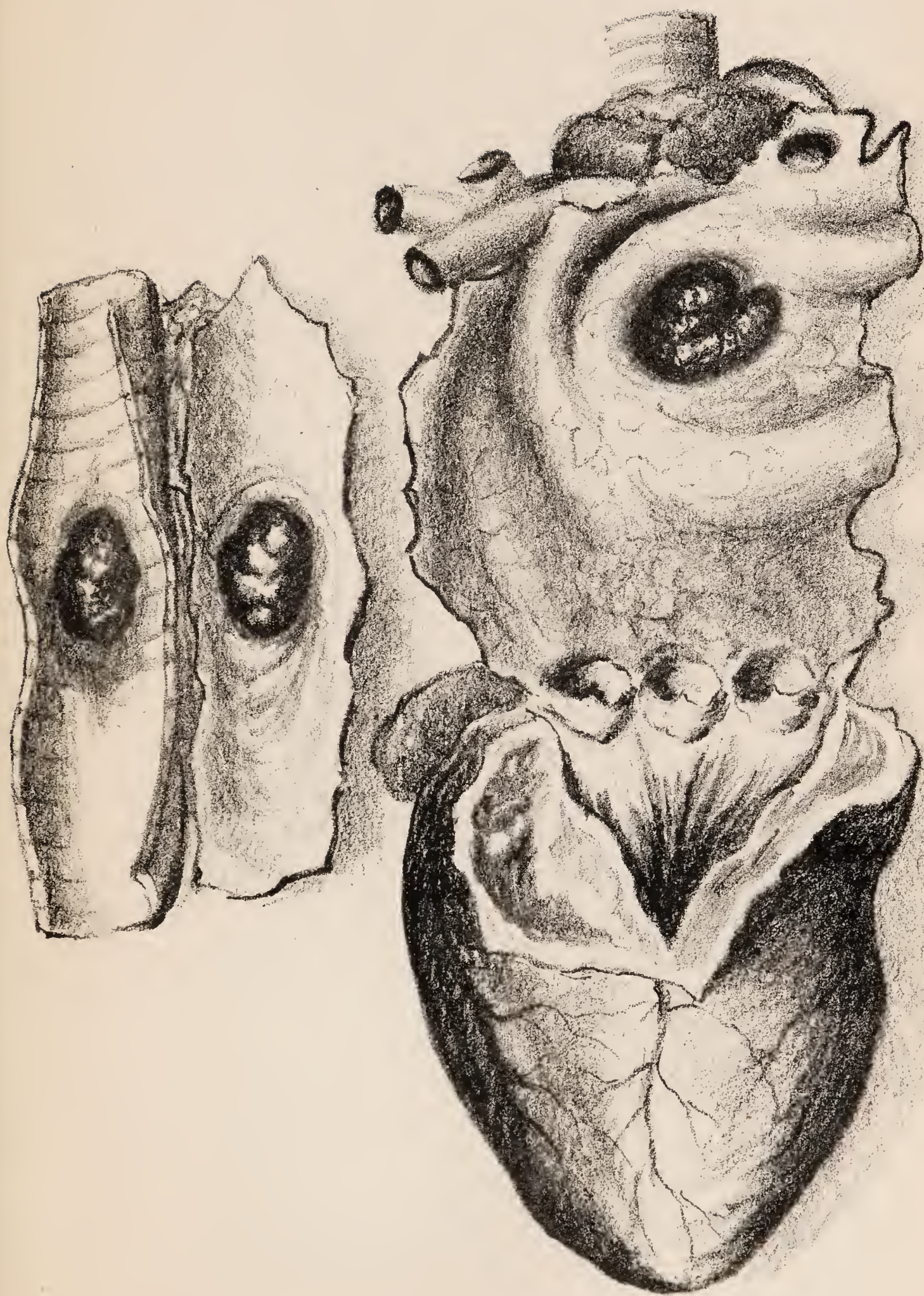
On July 2, during a fit of coughing, she became intensely cold and collapsed, but recovered after three hours; in the evening there was a second similar attack, and suspecting there had been hæmmorrhage into the œsophagus, I found on examination a gurgling as of much fluid in the intestines with distension and pain in the right hypochondrium.

July 3rd.—She seemed better again and swallowed more easily. She passed several motions in the day of blood. In the night she vomited suddenly a large quantity of blood and fell back dead.

*Post-mortem examination.*—There was no fluid in the pleuræ nor pericardium. The left lung was collapsed but healthy. The drawing shews a large clot projecting from the aneurismal sac into the cavity of a diseased aorta, somewhat rigid and mottled with deposit. The aneurism was about the size of an egg and closely pressed upon the trachea and œsophagus. On opening these a projection from the clot was seen bulging into each through a sloughy opening, the mucous membrane around that in the trachea being in a state of green decomposition.

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# REPORT ON ETHIDENE DICHLORIDE AS AN ANÆSTHETIC.

BY THE MEDICAL SUPERINTENDENT.

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ETHIDENE DICHLORIDE was administered as an anæsthetic in twenty-eight cases taken at random. Two preparations were used, one of a specific gravity of 1·220, which quickly evaporated when a small quantity was poured on the hand; the other of a specific gravity of 1·264, which was apparently less pure, as it did not evaporate so quickly nor so completely as the first, and left an oily residuum. The action of both preparations was nearly the same, both in the production of narcosis and in their after effects.

In those cases in which this anæsthetic was administered it was observed that the noisy struggling stage was almost absent. In most cases the patients went under without any struggling, and what little struggling appeared was of a purposeless character and of very short duration.

The pulse was affected as follows:—At first it was slightly accelerated and increased in force. Then, when the patient was almost unconscious and muscular relaxation had appeared, the pulse decreased in frequency to about the same number of beats per minute as it was before the operation and became softer. The colour of the patient remained very good throughout.

Respiration was affected in a somewhat similar manner, at first becoming more frequent and then slower, remaining throughout narcosis at about its normal frequency. As a rule, there was no stertor.

The pupils were unaffected, at least in all the cases except three. It was noticed that, at first, they became somewhat dilated, and then, later on, returned to about their normal size. In two of the three exceptions the pupils were considerably contracted, and remained so throughout the period of narcosis. In the third case the pupils, at first, became a little dilated and then re-contracted. Later on, when the anæsthetic had been administered for about fifteen minutes, and about 4 drs. had been given, the pupils became widely dilated. In this case there was considerable stertor, pallor appeared, the respiration became very weak and shallow, and the pulse became almost imperceptible at the wrist. Artificial respiration for about five minutes restored the patient. The pupils were then noticed to be about normal in size.

It was observed that, in the administration of ethidene, anæsthesia and muscular relaxation came on in the following order:—At first, insensibility of the skin of the trunk and extremities; then muscular relaxation of the upper extremities, then of the lower; by this time the skin of the temples was insensible; insensibility of the sclerotic conjunctiva followed next; and, last of all, the corneal conjunctiva became insensible. When the patients were fully under the influence of the anæsthetic the pupils were noticed to be inactive.

Once unconscious, the patients required very little of the anæsthetic to keep them thoroughly under. They took some time in coming round, but came to very



quietly. Vomiting was a very frequent and very distressing sequel. This, perhaps, was due to impurity of the ethidene employed. It occurred in 53 per cent. of the cases. Subsequent headache, too, was frequent and severe, though, on the whole, not so much so as in those cases in which chloroform was the anæsthetic employed.

The advantages of ethidene over chloroform seem to be that it is not a heart depressant, and that the noisy struggling stage is absent or nearly so. A disadvantage is that its after effects are more unpleasant, though, probably if a pure preparation were used, the vomiting and headache would be less distressing.

Comparative statements of the amount of anæsthetic required to produce and to keep up for twenty minutes a state of narcosis, and also of the occurrence of vomiting, are subjoined for the last 303 cases in which anæsthetics were administered. These refer to 152 cases in which a mixture of one part of chloroform to three of ether was used, 112 cases of pure chloroform, 28 of ethidene dichloride, and 11 of pure ether.

*Average amount (approximately) to produce complete narcosis :—*

Chloroform, one drachm and a half.

Chloroform and ether mixture, two to three drachms.

Ethidene dichloride, one drachm and a half.

Ether, could not be ascertained.

*Average amount to keep up narcosis for twenty minutes :—*

Chloroform,  $2\frac{1}{2}$  drachms.

Chloroform and ether mixed,  $3\frac{1}{2}$ —4 drachms.

Ethidene dichloride, over 3 drachms.

Ether, about 10 drachms.

*Occurrence of Vomiting :—*

Chloroform (pure), 112 times, vomiting in 30 cases,  
26·07 per cent.

Chloroform and ether (1 to 3), 152 times, vomiting in  
26 cases, 17·11 per cent.

Ethidene dichloride, 28 times, vomiting in 15 cases,  
53·57 per cent.

Ether (pure), 11 times, vomiting in 7 cases, 63·63 per  
cent.

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## A COMPLICATED CASE OF HERNIOTOMY.

BY MR. GREIG SMITH.

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A. B., æt. 18, labourer. 20 days.

The following case of herniotomy presents so many features of interest that it deserves to be recorded at some length. The hernia was into a patent vaginal process, and, besides two constrictions in the sac, one at the orifice of the vaginal process, the other at the junction of the funicular and testicular portions, there was constriction by a round fibrous cord inside the abdominal cavity.

The patient, a healthy, well nourished young man, had an inguinal hernia for two years, of small size, and reducible. The swelling had come on suddenly at first, and had slowly increased in size. He had not worn a truss. Three days before admission he had been working very hard turning a windlass, and at night he discovered that his rupture had become larger, and very painful, and that he could not, in spite of vigorous efforts, return it. His bowels had moved that morning.

Thirty hours after he found the hernia to be irreducible he came to the Infirmary. He complained of great pain and tenderness in the tumour and in the right iliac region (the tumour being on the right side). The hernial tumour was very hard and tense, gave no impulse on coughing,



and presented a marked hour-glass construction midway between its origin from the abdominal wall and its fundus. In the posterior aspect of the lower half of the tumour the testicle was imbedded, and on examination by transmitted light this lower portion was found to be translucent. It was tapped with a fine Southey's trocar inserted obliquely, and clear crystalline fluid flowed. The hernial contents distinctly diminished in size. Taxis was very gently tried, but without effect. Herniotomy was then performed in the usual way. On opening the sac a little pink serum was seen, very different in colour from the fluid removed on tapping. The constriction above the lower half of the tumour having been divided, the bowel, which occupied the cavity of the tunica vaginalis, sprang out of the wound, and rolled over the groin. A second constriction at the neck of the sac was divided, and more intestine bulged out. There being still some constriction at the internal abdominal ring, this also was divided. On making the last incision, more bowel and a mass of omentum rolled out. The groin, scrotum, and upper part of the thigh were now covered with coils of dark, ecchymosed intestine. I carefully washed, for the second time, all the parts on which the intestines rested with carbolic lotion, and turned on a second spray. I carried my finger through the inguinal canal into the abdominal cavity, felt all over the intestine, and around the margins of the abdominal opening, concluded that there was no further constriction, and proceeded to return the bowel. As soon as I returned it, however, it recoiled. A further and more careful examination by one of my colleagues and myself still revealed no constriction. Though there was not much intra-abdominal tension, yet the coils of bowel outside the abdomen were very tense with fluid. I there-

fore inserted a fine Southey's trocar obliquely into the bowel, and *clear pellucid fluid* flowed. The bowels became flaccid, but, in a few moments, became as tense as ever again, and still could not, by using what seemed a justifiable amount of force, be returned. I concluded that there must be some constriction inside the abdomen, and followed the bowel carefully upwards with two fingers as far as the promontory of the sacrum, and there a stricture was found. A hard, round, fibrous cord, as thick as a surgical probe, surrounded and constricted the double coil of strangulated intestine which had been giving all the trouble. With some difficulty, after enlarging the abdominal wound, it was brought within the reach of a knife by the forefinger of the left hand, and divided. On division of this cord, the distended bowel at once collapsed and was easily returned.

The subsequent history of the case is absolutely uneventful. On the second day the temperature was normal and continued so. There was no constitutional disturbance. At the end of a fortnight the patient expressed himself as feeling quite well, and wanted to get up and go to work. It was thought wise, however, to keep him in bed for a week longer.

Noteworthy features in the case are :—

1. An intestinal hernia had existed in the upper (funicular) portion of a patent vaginal process, and had forced its way through the ring which usually exists between this upper portion and the lower portion, or tunica vaginalis testis. Inside the latter cavity was the chief bulk of the hernia.
2. The globular lower portion of the hernial tumour had all the physical characters of a hydrocele of the tunica vaginalis;—even translucency existed.



Above this tumour the constriction was so tight that the neck might have passed as a thickened spermatic cord. The clear serum which flowed on tapping might have been taken as further evidence of the existence of hydrocele: yet this serum undoubtedly came from the inside of the bowel, and was a secretion of the intestinal glands.

3. The constriction in the sac at the internal abdominal ring was probably caused by the thickened and contracting abdominal orifice of the vaginal process of the peritoneum.
4. The most puzzling complication was the existence of a stricture inside the abdominal cavity. The coils of bowel which leapt out of the wound were all strangulated by this band. The intestine was darkest in colour where it lay in the tunica vaginalis, was less dark where it lay in the funicular portion of the sac, and was only pink and injected between the abdominal orifice and the intra-abdominal constriction. The bowels, which were distended with clear intestinal secretion, at once collapsed when the band was divided. What was the nature of this band, and to what structures it was adherent, I cannot say. It is just possible that it may have been the effect of a localised peritonitis set up by the hernia.

The lessons to be learnt are, that, if the symptoms of strangulated hernia are present, we are not to conclude that the presence of a clear translucent fluid inside the tunica vaginalis is proof of the existence of hydrocele; and that, if more than the ordinary difficulty in returning a distended bowel is experienced, we are not to rest satisfied with having divided all the ordinary constrictions.



Such complications as were found in the above case must be extremely rare; the possibility of this occurrence is all the more instructive.

*Note.*—Mr. Augustin Prichard, on my having described to him the peculiarities of the above case, very kindly supplied me with notes of a complicated case of congenital hernia, on which he operated in 1860. In this case the condition was verified by *post mortem* examination.

G. W., æt. 46, had always had a tumour in the right groin. When seen it had been strangulated 48 hours. During these 48 hours it had been twice reduced by the medical attendant, but without relief to the symptoms. Mr. Prichard operated, and came down on a thick sac, which he opened and found nothing. The finger passed up through the ring and intestine was felt above; but the finger did not pass freely into the abdomen. A large quantity of fluid escaped, and there was considerable foetor with it. A substance like a cord was found in the sac. No relief followed. The patient lingered till the morning after operation, when he died quietly, having suffered but little pain since the operation. Vomiting recurred a short time before death.

At the *post mortem* it was found that the hernia had been a congenital one, and had been reduced *en masse*, leaving a portion of sac behind. There was complete strangulation by the inverted neck of the sac, and some general peritonitis, with fluid and partial adhesions.

The tendency of a congenital hernia to become reduced *en bloc* is well known; and the possibility of the neck of the vaginal process becoming adherent to the promontory of the sacrum, and acting as the intra-abdominal constricting band suggested itself to me, and

was rejected. That this band had no connection with the hernial structures proper is evident from the following considerations. I clearly saw and divided the only two strictures that could exist in the vaginal sac, and a third still remained. This third band had no peritoneal covering attached to it, but was a simple, round, firm, fibrous cord, which could be hooked up under the finger, and was firmly adherent to some structure near the promontory of the sacrum. My fingers, introduced through the abdominal opening, moved freely about among the bowels, and it was easy enough to settle that the intra-abdominal band had no anatomical connection with the sac of the hernia. At least three feet of bowel passed under this band, and were strangulated by it. I think there can be no doubt that this case must be regarded as one in which internal strangulation was superadded to strangulation by the ordinary means. So far as I know, the case is unique.

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## SURGICAL REPORT.\*

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THE Bristol Royal Infirmary, built rather more than a century ago, was originally a long H-shaped building of three stories. On the ground floor were casualty wards, House Surgeons', Pupils' and Committee rooms, with various offices; underground were the kitchens and bake-house; whilst the first and second floors were occupied by wards. Ventilation was carried out by windows alone, some wards being ventilated into the external air on two sides, while others communicated on one side with the central corridor which ran from end to end of the building.

In 1852 a wing consisting of a museum and chapel was built at one end of the main building; and in 1869 a corresponding wing containing two wards was erected at the other end. The Infirmary was now converted from its original plan into three sides of a square, which enclosed the out-patient buildings. Thus the free circulation of air at the back of the building was interfered with; at the same time that the emanations from the out-patients had every opportunity of passing through the open windows into the wards.

\* The statistical tables up to 1876 were compiled, and the substance of this paper written, by the late Mr. Tibbits. For the rest the surgical editor is responsible.



Soon after this the Faculty noticed that the patients did not progress in so satisfactory a manner as formerly; that convalescence was slow, that erysipelas was getting a firm footing, and that pyæmia began to appear with alarming frequency. In the year 1873 the deaths from pyæmia numbered 14; in 1874 they were 33; and, in the first eight months of 1875, 16 more were added to the list. This does not include any of the deaths from low forms of peritonitis after hernia operations—which were, during this time, almost invariably fatal—and of cystitis which constantly increased the fatality of operations upon the urinary organs. Scarcely ever was a ward free from erysipelas; and at last a healthy wound was so rarely seen as to become an object of general interest.

All the while more than ordinary care had been paid to sanitary details. The walls of the wards were frequently distempered and re-coloured; the floors were regularly washed with solutions of carbolic acid; the closets were flushed with disinfectants, and the bedding materials properly attended to. But all seemed to be without beneficial effect.

In February, 1874, the Faculty forwarded to the Managing Committee a report describing the bad sanitary condition of the house, and strongly recommending a thorough renovation of the building. Acting upon this report and on the advice of an architect skilled in hospital construction, the Committee in September, 1875, secured temporary premises for the treatment of the most severe cases, and closed the Infirmary buildings for repairs. The alterations carried out were shortly as follow:—Removing all the old woodwork and flooring, and in some cases the laths of the ceiling; replacing the packing

between the ceiling and floors with asphalt; accurately “tonguing” the floors as they were relaid; scraping off all the old plaster from the walls and replacing it with new; altering all the windows, and introducing what is known as the “Middlesex sash;” introducing into the corridors and some of the wards Tobin’s ventilating tubes; placing Sherringham ventilators in other wards; removing all the old drain pipes, and laying down a perfectly new system of drainage; in placing the lavatories and water-closets in projecting turrets completely shut off from the wards, and ventilated into the open air; and replacing the old well water supply by a thorough service from the Water Company, the closets being served through a system of pipes distinct from that of the rest of the house. Lastly, a wooden house, containing twelve beds, was erected at a distance of sixty yards from the main building for the reception of such cases of infectious disease as might originate in the house. The carrying out of these and other alterations extended over a period of twelve months and cost over £20,000. The Infirmary was open for the reception of patients in October, 1876.

The rates of mortality before and after the alterations will now be considered. The period selected prior to the alterations—from 1861 to 1875—may be divided into two:—

1. A period of eight years preceding 1869, when the last new wing was built, which might be supposed to interfere with the free circulation of air round the building.

2. The period from 1869 to the time when the Infirmary was closed in September, 1875.

First as regards the general death-rate from all causes.

### GENERAL DEATH-RATE PER CENT.

Year.	Mortality per cent.	Year.	Mortality per cent.
1861	4.47	1869	4.93
1862	4.93	1870	5.23
1863	5.06	1871	5.93
1864	6.20	1872	7.35
1865	5.08	1873	6.62
1866	4.71	1874	6.72
1867	5.13	1875 9 mos.	6.06
1868	4.60		
Average for 8 years ... }	5.03	Average for 7 years ... }	6.07

In the first period the general mortality stood at 5.03 per cent., in the second period at 6.07, or an increase of 1 per cent. And, as between 2,500 and 3,000 patients annually pass through our wards, this means an aggregate increase of 25 or 30 deaths per annum. Looking more closely into these figures, it will be seen that only once in the first period did the mortality rise above 6 per cent., whilst for the four years preceding the closing of the Infirmary it never fell below this figure.

Dissecting these figures, and dividing them into classes, shews that the increased mortality was not strictly confined to any one department or particular class of cases.



MEDICAL AND SURGICAL DEATH-RATES FROM 1865 TO  
1868 INCLUSIVE.

Year.	Medical per cent.	Surgical per cent.
1865	6.63	3.88
1866	6.70	3.11
1867	7.36	3.29
1868	6.94	2.72
Average ...	6.90	3.27

MEDICAL AND SURGICAL DEATH-RATES FROM 1869 TO  
1875 INCLUSIVE.

Year.	Medical per cent.	Surgical per cent.
1869	6.73	3.47
1870	6.83	4.02
1871	7.23	4.01
1872	9.57	5.28
1873	10.00	3.59
1874	9.24	4.70
1875 9 mos.	8.21	5.19
Average ...	8.26	4.25

In the four years preceding 1869 the general medical death-rate averaged 6.90 per cent., and in the seven succeeding years this rose to 8.26 per cent. During the

same periods the general surgical death-rate rose from an average of 3·27 to 4·25.\*

Turning from the general rate of mortality to that of a special class most likely to be influenced by their surroundings, viz., those requiring surgical operation, we get the following figures :—

DEATH-RATE PER CENT. AFTER ALL OPERATIONS.

Year.	Mortality per cent.	Year.	Mortality per cent.
1861	3·78	1869	7·36
1862	7·79	1870	15·38
1863	5·59	1871	10·42
1864	7·33	1872	15·33
1865	7·13	1873	15·07
1866	3·87	1874	12·89
1867	7·83	1875 9 mos.	15·73
1868	3·24		
Average mor- tality p. c.	5·74	Average mor- tality p. c.	12·93

\* When the late Mr. Tibbits wrote the above he was convinced that he had made out a clear case of increasing mortality from bad hygiene. He would have been surprised to find that, in spite of our improved sanitary conditions, our death-rate, medical as well as surgical, was still on the increase. The surgical mortality in '78 was 4·67, in '79 was 4·79; the medical mortality in these years was 9·83 and 9·81. And this general increase exists in spite of undoubted and striking improvements in the death-rate from preventable hospital diseases. In fact no better example could be got of the fallacy of dealing with hospital mortality by bare statistics, which consist simply of numbers without super-added particulars. The manifold ways in which such statistics may mislead are pointed out in the masterly Report on the Hospitals of the United Kingdom, by Bristowe and Holmes, in the Sixth Report of the Medical Officer of the Privy Council, and need not be here repeated. As they justly remark, a high hospital mortality is more frequently evidence of superior usefulness than of defective sanitation. In our own case it would be possible to shew that while the mortality after operations and from hospital diseases has greatly diminished, the general death-rate has increased chiefly from the greater gravity of the cases admitted, arising from the fact that, though the population has immensely increased within the last twenty years, there has been no corresponding increase in our Infirmary accommodation.—J. G. S.

Thus it will be seen that in the eight years preceding 1869 the death-rate after operations of all sorts averaged 5·74 per cent., whilst from 1868 to 1875 this had risen to 12·93 per cent.,—in fact more than doubled. The grave significance of these figures is sufficiently clear, when it is noted that nearly 200 operations that might lead to death are performed annually in our Infirmary.

Again, from the general class of surgical operations we may select cases of amputation of the thigh and leg for especial comparison. These, it will be admitted, belong to a class of cases which are not materially influenced by the skill of individual operators.

MORTALITY PER CENT. AFTER AMPUTATIONS OF THE  
THIGH AND LEG.

Year.	Number of Cases.	Year.	Number of Cases.
1861	8	1869	18
1862	9	1870	15
1863	4	1871	18
1864	14	1872	17
1865	12	1873	19
1866	12	1874	13
1867	13	1875 9 mos.	7
1868	14		
Total ...	86	Total ...	107

After the 86 amputations of the thigh and leg, from 1861 to 1868, there were 14 deaths, a mortality per cent. of 16·27.

After the 107 amputations of thigh and leg, from 1869



to 1875, there were 41 deaths, a mortality per cent. of 36·75.\*

The records are not sufficiently complete to permit of the compilation of a trustworthy table shewing the mortality from pyæmia and erysipelas. It may be stated, however, that, in 1873, 14 deaths were recorded from pyæmia or erysipelas; in 1874, 23 cases, and in the first eight and a half months of 1875, 16 cases were added to the list.

Now, what has been the effect on our death-rate, firstly, on removal to temporary premises in 1875-76, and, secondly, after re-occupation of the renovated Infirmary from 1876-79 inclusive?

In the temporary premises, ordinary dwelling houses with no special adaptations for medical or surgical purposes, there was accommodation for 79 patients. Only the most urgent cases were admitted, and chronic cases, and such as had only clinical or pathological interest were entirely excluded. In these buildings 88 major operations were performed, and there were 10 deaths, a mortality of 11·38 per cent. Four of these deaths were due to tracheotomy for croup and diphtheria, and two to trephining for severe compound fractures of the skull with laceration of the brain. The only 10 amputations of the thigh and leg (one a double amputation) were successful. There were three deaths from pyæmia or erysipelas, one, a herniotomy, doubtful.

During the three years and two months of our occu-

\* Among Metropolitan Hospitals, the lowest mortality after thigh and leg amputations, during the same period, was in St. Bartholomew's, with 20 per cent.; in St. George's Hospital, during 1877-78, the mortality was 21 per cent. after the same operations; in Guy's, during 1877, 25 per cent. The mortality in other Hospitals which publish reports, comes more nearly to that of Bristol at its worst. During 1877-78-79 there were 53 amputations of the thigh and leg in the Bristol Infirmary, with 5 deaths, a percentage of 9·4.—J. G. S.

pation of the restored Infirmary, we have had no deaths from pyæmia or erysipelas. Indeed, we have hardly seen a genuine case of either disease. An out-patient with a putrid glandular abscess in the chin, had suppuration in the knee joint, and recovered with a movable joint. Another patient, in a neighbouring bed, with a syphitic ulcer in the popliteal space, had suppuration of the knee joint, and, after amputation of the thigh, also recovered. Two very old men died of asthenia after amputation of the leg and arm respectively. Such is our experience of pyæmia. The only case of genuine erysipelas which has occurred in these three years was in a case of removal of a large cancer of the vulva, where the wounds, in spite of every precaution, came into frequent contact with both urine and foeces. A few instances, not more than half a dozen, of mild erythema, which passed off in a day or two, have been observed. Under the open, or cold water methods of dressing wounds, these last mentioned instances of erythematous erysipelas would probably have been spoken of as the ordinary inflammatory blush which was seen on most wounds; where the Listerian method is pursued the existence of such a blush is taken as evidence of septicism. In no antiseptic case has such a blush ever been observed.

A more positive result than the absence of hospital diseases has been observed in the rapid healing of wounds and the absence of constitutional disturbance after operations. When it is said that primary healing is the rule and suppuration the exception, that constitutional disturbance is so slight that the ordinary meat diet is rarely changed; and that stimulants or tonics are scarcely ever demanded after operation, our satisfactory position will be more readily understood.

How much of this is due to the antiseptic method of



Lister, which is pursued by all the surgeons ; and how much to the hygienic condition of the building it is impossible to say. That Listerism, apart altogether from general hygienic improvements, is capable, single handed, of doing away with most hospital diseases there is now no room to doubt. But it is impossible to start with all wounds aseptic ; and, through its efforts at conservation of limbs after compound fractures, the antiseptic method presents greater opportunities for pyæmia than other methods do. There is always a certain amount of septic material in the atmosphere of a hospital ; and though the danger of infection be reduced to a minimum, it is still there, and must be guarded against just as stringently as if it existed to saturation. This truth is all the more important in our case, because the hospital is always too much crowded, and the cubic space to each patient dangerously small, being considerably under 1,000 feet.

In 1877 there were 21 amputations of the thigh and leg with 3 deaths. There were 5 amputations of the thigh with 1 death, in a man aged 67, who, after one thigh had been amputated for gangrene, succumbed to gangrene of the other leg. In 16 leg amputations there were 3 deaths, one from thrombosis of the pulmonary artery ; one in a man aged 68, from spreading gangrene ; the third died on the operation table, from a general crush, and would, if he had rallied, have had his other leg amputated. There were 4 amputations of the arm (two shoulder), and 5 of the forearm, with no death. There were 22 excisions of important tumours (7 excisions of mamma) with no death. There were 7 herniotomies, with 1 death, in a baby with convulsions.

Tabular statements of our chief operations and their results during 1878-79 are given in the following tables.



No.	Age.	Sex.	Variety of Hernia.	Duration of Hernia.	Duration of Strangulation.	Vomiting: Duration and Character.	Treatment prior to Admission.	Operation and Remarks.	Progress of Case and Remarks.	Result.	Days in House.
1	54	F.	Femoral.	Recent.	72 hours.	Bilious 48 hours, ster- coraceous 12 hours.	None.	Sac not opened. Stricture at femoral ring. Bowel reduced.	Tedious convalescence. Tympanites troublesome. Patient in bad health before strangulation took place.	C.	58
2	46	M.	Very large scrotal, reducible.	10 years.	24 hours.	Bilious 14 hours.	Taxis.	Sac opened. Stricture at neck of sac. Much pink serum. Bowel chocolate coloured. Hernia very tender.	Subacute peritonitis. Vomited three times after operation. Wound healed rapidly, but tedious recovery.	C.	52
3	66	M.	Femoral, irreducible.	25 years.	38 hours.	Bilious 24 hours, ster- coraceous 3 hours.	Taxis.	Sac opened. Stricture at neck of sac, very thick. Chiefly omentum. Bowel very dark. Some pink serum. Hernia not reduced.	Chronic bronchitis and emphysema with very troublesome cough. Wound healed kindly, but tardy convalescence. Transferred to medical ward.	C.	104
4	73	F.	Femoral, reducible.	Recent.	70 hours.	Bilious 48 hours, ster- coraceous 4 hours.	None.	Sac not opened. Stricture at femoral ring. Bowel reduced.	Patient a drunken old woman, had delirium tremens. Primary union in wound and excellent recovery.	C.	33
5	38	M.	Scrotal, reducible.	10 years.	32 hours.	Bilious 24 hours.	Taxis.	Sac not opened. Stricture at internal abdominal ring. Bowel returned.	Primary union. Excellent recovery. No symptoms.	C.	23
6	78	M.	Very large scrotal, reducible.	40 years.	7 days.	Bilious 4 days, ster- coraceous 3 days.	Taxis by patient.	Sac opened. Stricture at neck of sac and abdominal ring. Bowel very dark, and ecchymosed, but glistening, returned. Much pink serum and lymph.	Pulse scarcely perceptible during operation. Patient never rallied, and died ten hours after admission.	D.	1

## CASES OF HERNIOTOMY—continued.

No.	Age.	Sex.	Variety of Hernia.	Duration of Hernia.	Duration of Strangulation.	Vomiting : Duration and Character.	Treatment prior to Admission	Operation and Remarks.	Progress of Case and Remarks.	Result.	Days in House.
7	55	F.	Femoral reducible.	2 years.	50 hours.	Stercoraceous 3 hours.	Taxis.	Sac opened. Strictures at neck of sac and femoral ring. Bowel, ecchymosed and purple, returned. Clear serum.	Primary union. Uninterrupted recovery.	C.	12
8	39	F.	Inguinal reducible.	12 years.	5 days.	Bilious.	None.	Sac opened. Tense stricture at int. abdominal ring. Very dark omentum traversed by fibrous bands ; partly returned.	Patient had advanced amyloid disease of kidneys and liver ; albuminuria $\frac{4}{5}$ ; subcutaneous cedema. Wound healed kindly.	D.	7
9	72	M.	Inguinal oblique.	Recent.	61 hours.	Bilious 50 hours, stercoraceous during operation.	Taxis.	Sac not opened. Stricture at int. abdominal ring. Very tight. Bowel returned of itself on division of stricture.	Patient was suffering from an attack of bronchitis when admitted ; the hernia came down during a fit of coughing. Was in very low condition during operation, and did not rally.	D.	1
10	48	F.	Femoral irreducible.	14 years.	6 days.	Bilious 3 days, stercoraceous 2 days.	None.	Sac opened. Stricture at femoral ring. Bowel and omentum. Part of omentum cut off, bowel returned.	Wound healed by primary union. Death from enteritis and peritonitis.	D.	5
11	43	M.	Scrotal reducible.	25 years.	28 hours.	Bilious 12 hours.	Taxis by patient.	Sac opened. Strictures at abdominal ring and neck of sack. Ecchymosed bowel. Brown serum. Cutaneous bruising from taxis.	Patient had phthisis, with night sweats and severe cough. No symptoms from operation. Wound healed at second dressing. Went out wearing truss.	C.	18



No.	Age.	Sex.	Variety of Hernia.	Duration of Hernia.	Duration of Strangulation.	Vomiting: Duration and Character.	Treatment prior to Admission.	Operation and Remarks.	Progress of Case and Remarks.	Result.	Days in House.
12	61	F.	Femoral reducible.	30 years.	20 hours.	Bilious 14 hours.	Taxis. Ice.	Sac opened. Stricture at femoral ring. Bowel (very dark) and omentum both returned.	Wound became septic and suppurated. Difficulty in getting bowels opened. Very irritable patient.	C.	21
13	50	F.	Femoral reducible.	20 years.	76 hours.	Bilious 3 days.	None.	Sac opened. Stricture at femoral ring. Bowel adherent to sac at parts, and traversed by fibrous rings. Tinged serum. Hernia very large. Bowel returned.	Vomiting continued for six days after operation, but never became stercoraceous. Bowels moved with difficulty. Sickness disappeared and a good recovery was made. Primary healing of wound.	C.	30
14	60	F.	Femoral.	Recent.	43 hours.	Bilious 43 hours.	Taxis.	Sac not opened. Stricture at femoral ring. Bowel returned.	Patient was very feeble and decrepid, and had albuminuria, fr. cirrhosis of kidney. Peritonitis and pinhole perforation of gut. Vomited four times after operation. Never passed motion or flatus. P.M.E. revealed extensive enteritis, almost gangrene of gut, and local peritonitis, but no perforation or stricture.	D.	3
15	43	M.	Inguinal reducible.	2 years.	29 hours.	Bilious 20 hours, stercoraceous 4 hours.	Prolonged taxis by patient.	Sac opened. Tight constriction at int. abdominal ring. Bands near sac. Much dark serum. Bowel very dark and ecchymosed, returned.		D.	2
16	22	M.	Inguinal reducible.	10 days.	36 hours.	Bilious 23 hours.	Taxis by patient.	Sac opened. Stricture at int. abdominal ring. Small knuckle of congested bowel returned. Hydrocele of cord, large and tense, laid open.	Excellent recovery. Wound skinned over in a week. Wearing truss on twelfth day.	C.	12



## CASES OF HERNIOTOMY—continued.

No.	Age.	Sex.	Variety of Hernia.	Duration of Hernia.	Duration of Strangulation.	Vomiting: Duration and Character.	Treatment prior to Admission.	Operation and Remarks.	Progress of Case and Remarks.	Result.	Days in House.
17	70	M.	Scrotal reducible.	12 years.	32 hours.	Bilious and stercoraceous.	Taxis by patient.	Sac opened. Stricture at neck of sac and abdominal ring. Bowel very dark, opened, and artificial anus.	Favourable progress. Artificial anus slowly contracting. Fæces passed only occasionally through opening.	C.	40
18	50	F.	Femoral reducible.	8 years.	3 days.	Bilious 24 hours, stercoraceous 12 hours.	None.	Sac opened. Strictures at femoral ring and neck of sac. Pink serum. Omentum and bowel both returned.	Excellent recovery. Wound quite healed at second dressing. Wearing truss on fourteenth day.	C.	16
19	51	M.	Scrotal reducible.	1 year.	32 hours.	Bilious 48 hours.	Taxis by patient.	Sac opened. Stricture at abdominal ring. Bowel and omentum glued to sac. Much pink serum.	Favourable progress. Vomited twice after operation.	C.	24
20	41	F.	Femoral reducible.	3 mos.	6 days.	Stercoraceous 48 hours.	None.	Sac opened. Stricture at femoral ring. Bowels very dark, but glistening. Dark serum, and flakes of adherent lymph.	Patient was in very exhausted condition, and never rallied after operation. Vomiting ceased after operation. P.M.E. revealed enteritis and pulmonary congestion.	D.	4
21	48	M.	Femoral reducible.	10 years.	6 days.	Stercoraceous 5 days.	Taxis by patient.	Sac opened. Stricture at femoral ring. Gut perforated and gangrenous. Fæces in abdominal cavity. Artificial anus.	Vomiting had ceased 20 hours before admission, and patient was in state of profound collapse during operation, from which he never rallied, and died five hours afterwards.	D.	

No.	Age.	Sex.	Variety of Hernia.	Duration of Hernia.	Duration of Strangulation.	Vomiting: Duration and Character.	Treatment prior to Admission.	Operation and Remarks.	Progress of Case and Remarks.	Result.	Days in House.
22	18	M.	Infantile reducible.	2 years.	36 hours.	Bilious.	None.	Sac opened, found to be vaginal process. Strictures at middle of sac, abdominal ring, and by round fibrous cord inside abdominal cavity.	Excellent recovery. No bad symptoms.	C.	20
23	60	F.	Femoral.	Recent.	1 wk.	Bilious and stercoraceous 4 days.	None.	Sac opened. Omentum and bowel (very dark) both returned.	Sickness stopped. Great exhaustion; died in 16 hours. P.M.E. revealed enteritis in three feet of intestine.	D.	

During the years 1878-79 there were 23 operations of herniotomy with 9 deaths (in 1877 there were 7 operations with 1 death).

An examination of the points noted reveals the following:—

*Age.*—The youngest was 18, the oldest 78. Arranged in periods of ten years they are,—

Between 10 and 20 cases				1 deaths 0			
“	20	“	30	“	1	“	0
“	30	“	40	“	2	“	1
“	40	“	50	“	6	“	4
“	50	“	60	“	5	“	0
“	60	“	70	“	4	“	2
“	70	“	80	“	4	“	2

*Sex.*—Female, 11, 5 deaths; male, 12, 4 deaths.

*Variety of Hernia.*—Femoral 12—10 in females, 2 in males, 5 deaths; inguinal 11—1 in female, 10 in males, 4 deaths. Of femoral herniæ two were irreducible, all were reducible of the inguinal variety.

*Duration of Hernia.*—Recent to 40 years.

*Duration of Strangulation.*—20 hours to 7 days.

Arranged in periods of ten hours they are,—

Hours	20—30	cases	4	deaths	1
“	30—40	“	6	“	0
“	40—50	“	1	“	1
“	50—60	“	1	“	0
“	60	“	10	“	7

*Character of Vomit.*—Bilious alone, 10 cases, 2 deaths; bilious and stercoraceous, 13 cases, 7 deaths.

*Operation.*—Sac opened in 18 cases, 7 deaths; sac not opened in 5 cases, 2 deaths.



*Stay in the House.*—The average stay of those who recovered (excluding one case which was transferred to a medical ward for bronchitis) was 27·1 days. Among the shortest periods are 14, 16, 18, 12, 18, 12 days. Patients are sent out wearing trusses.

Nothing new is to be gathered from an examination of the causes of mortality. As usual no influence competes with that of prolonged strangulation. Not even old age competes with it. In seven of the nine deaths symptoms of strangulation had lasted for sixty hours and upwards; of the other two, one died from excessive use of the taxis, the other from advanced kidney disease.

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## AMPUTATIONS OF THIGH.

No.	Sex and Age.	Disease and Operation.	No. of Days in House.	Result.
1	M. 55	Crushed leg and knee. Other severe injuries. Modified circular.	6	Death.
2	F. 22	Carious disintegration of knee joint. Sinuses in flaps. Mid. $\frac{1}{3}$ . Circular, skin flaps.	69	Recovery.
3	M. 14	Extensive necrosis of tibia. Disorganisation of knee joint. Solid œdema of leg and thigh. Up $\frac{1}{3}$ . Skin flaps.	101	Recovery.
4	F. 17	Caries and osteo-myelitis of femur after excision of knee. Mid. $\frac{1}{3}$ . Skin flaps.	36	Recovery.
5	F. 15	Disorganised knee joint. Great wasting. Abscesses and sinuses. Mid. $\frac{1}{3}$ . Skin flaps.	48	Recovery.
6	M. 38	Caries of femur. Complete disorganisation of knee joint. Sinuses and abscesses. Mid. $\frac{1}{3}$ . Modified circular.	42	Recovery.
7	F. 13	Caries of femur after excision. Numerous sinuses. Mid. $\frac{1}{3}$ . Skin flaps.	34	Recovery.
8	F. 22	Disorganised knee joint. Caries of tibia. Sinuses. Low. $\frac{1}{3}$ . Skin flaps.	30	Recovery.
9	M. 39	Disorganised knee joint. Advanced phthisis. Done to relieve pain. Mid. $\frac{1}{3}$ . Flap.	36	Death.
10	M. 4	Caries of femur and tibia. Disorganised knee. Mid. $\frac{1}{3}$ . Flap.	29	Recovery.
11	F. 15	Acute necrosis of tibia. Hectic and wasting. Carden's method.	33	Recovery.
12	M. 29	Complete disorganisation of knee. Hectic and great wasting. Mid. $\frac{1}{3}$ . Skin flaps.	25	Recovery.
13	F. 24	Caries of tibia and femur after excision of knee joint. Mid. $\frac{1}{3}$ . Skin flaps. Excessive pain in stump afterwards, and secondary amputation.		Recovery.
14	M. 25	Distortion of limb after injury. Osteotomy. Hæmorrhage from bone. Amputation by skin flaps above condyles.	38	Recovery.

AMPUTATIONS OF THIGH—*continued.*

No.	Sex and Age.	Disease and Operation.	No. of Days in House.	Result.
15	M. 67	Disorganised knee joint. Abscesses. Low. $\frac{1}{3}$ . Skin flaps. Sanguineous oozing for a month.	40	Recovery.
16	F. 42	Suppurating knee joint. Caries of femur. Patient very stout. Skin flaps. Mid. $\frac{1}{3}$ .	37	Recovery.
17	F. 25	Acute suppuration in knee joint from acute pyæmia. Large abscess in thigh. Extensive bedsore. Mid. $\frac{1}{3}$ . Flap.	75	Recovery.

There were 17 amputations of the thigh, 1 primary and 16 for disease; 8 in females, 9 in males. There were 2 deaths, neither of which could be ascribed to the operation. One case was the primary amputation, in which there was found, at the *post mortem*, rupture of the liver. The other patient was dying from phthisis, and amputation was performed at his own request to relieve pain, and apparently prolonged life for a few weeks. The average stay in the house of those who recovered was 45 days. This period is over debited from the prolonged stay of three patients, one on account of bedsore existing before operation, another from excessive anæmia and general debility which existed on admission, and the third from an attack of acute bronchitis. The patients are sent out wearing artificial limbs supplied by the Infirmary.



## AMPUTATIONS OF LEG.

No.	Sex and Age.	Disease and Operation.	Days in House.	Result.
1	M. 11	Caries and necrosis of tibia. Disorganised ankle. Up. $\frac{1}{3}$ . Skin flaps.	35	Recovery.
2	M. 44	Compound comminuted fracture. Sloughing before operation. Up. $\frac{1}{3}$ . Imperfect flaps and subsequent sloughing.	76	Recovery.
3	M. 27	Burn by boiling lees. Up. $\frac{1}{3}$ . Imperfect flaps and sloughing.	73	Recovery.
4	M. 12	Extensive caries of tarsus and tibia. Disorganised ankle joint. Mid. $\frac{1}{3}$ . Posterior flap.	44	Recovery.
5	M. 27	Crush of both legs by railway wagon. Double amputation by skin flaps. Mid. and up. $\frac{1}{3}$ .	72	Recovery.
6	M. 63	Epithelioma of leg. Up. $\frac{1}{3}$ . Anterior flap.	42	Recovery.
7	M. 42	Crushed leg by machinery. Up. $\frac{1}{3}$ . Skin flaps.	29	Recovery.
8	M. 44	Compound comminuted fractures of both legs. Amputation at lower $\frac{1}{3}$ of one; delayed union in other.	193	Recovery.
9	M. 59	Extensive malignant disease of foot and leg. Patient very weak. Skin flaps. Up. $\frac{1}{3}$ . Death from gradual exhaustion.	44	Death.
10	M. 35	Keloid of stump after Chopart's amputation. Edema of leg. Posterior flap. Mid. $\frac{1}{3}$ .	27	Recovery.
11	M. 46	Epithelioma in stump after Chopart's amputation. Up. $\frac{1}{3}$ . Posterior flap.	24	Recovery.
12	M. 23	Caries of Tarsus. Syme's amputation.	34	Recovery.
13	M. 39	Crushed foot. Run over. Syme's amputation. Heel flap sloughed.	79	Recovery.
14	M. 22	Traumatic gangrene of foot. Cellulitis of leg. Syme's amputation.	32	Recovery.
15	M. 20	Caries of Tarsus. Sinuses. Syme's amputation.	49	Recovery.

There were 15 amputations of the leg, 5 primary, and 10 for disease or at some lengthened period after injury; all in males. There was one death, in the case of a broken down old man with malignant disease, six weeks after operation. There was one double amputation, primary. The average stay in the house was 57 days. One patient was detained nearly 200 days for delayed union in a compound fracture of the leg. There was sloughing of flaps after three primary amputations, but good stumps were obtained in all.

### AMPUTATIONS OF ARM.

No.	Sex and Age.	Disease and Operation.	Days in House.	Result.
1	F. 39	Limb crushed in railway accident. Fracture of other arm. Amputation at shoulder joint. Outside flap.	65	Recovery.
2	F. 4	Limb crushed by being run over. Amputation at shoulder joint. Skin flaps.	40	Recovery.
3	M. 41	Recurrent fibroid of arm and forearm. Up. $\frac{1}{4}$ . Skin flaps.	32	Recovery.
4	M. 34	Recurrent sarcoma of arm. Flap. Up. $\frac{1}{3}$ .	27	Recovery.

## AMPUTATIONS OF FOREARM.

No.	Sex and Age.	Remarks.	Days in House.	Result.
1	M. 40	Hand and forearm crushed by machinery. Up. $\frac{1}{3}$ .	24	Recovery.
2	M. 28	Crushed hand and wrist. Amputation at mid. $\frac{1}{3}$ after 3 weeks.	15	Nearly well.
3	M. 13	Crushed hand and wrist. Skin flaps. Low. $\frac{1}{3}$ .	26	Recovery.
4	M. 17	Crushed hand. Amputation through wrist.	6	Healing.
5	M. 17	Crushed hand and forearm. Up. $\frac{1}{4}$ . Skin flaps.	17	Recovery.
6	M. 60	Epithelioma of hand and forearm. Mid. $\frac{1}{3}$ .	10	Death.
7	M. 55	Disorganised wrist joint. Muscle flaps. Mid. $\frac{1}{3}$ .	53	Recovery.

There were 4 amputations of the arm, 2 primary in females at the shoulder joint, 2 in males for malignant disease. There was no death. The average stay in the house was 41 days.

There were 7 amputations of the forearm, all in males; 5 primary, 2 for disease. There was one death, in a feeble old man with malignant disease, from exhaustion.



## EXCISIONS OF TUMOURS.

No.	Age.	Remarks.	No. of Days in House.
EXCISIONS OF MAMMARY GLAND FOR CANCER OR OTHER MALIGNANT DISEASE.			
1	43	Axillary glands removed.	41
2	60	Extensive implication of skin, incipient ulceration.	34
3	39	No peculiarity.	29
4	35	Tumour large.	12
5	52	Axillary glands removed. Recurrence in eight months.	29
6	30	No peculiarity.	16
7	63	Small axillary gland removed.	20
8	48	Extensive implication of skin.	44
9	53	Wound suppurated. Recurrence in three months.	45
10	54	Thymol as antiseptic. Wound suppurated.	41
11	42	Thymol as antiseptic. Wound suppurated.	35
12	72	Patient very stout. Extensive wound.	17
13	24	No peculiarity.	8
14	38	Fibro-sarcoma.	7

## EXCISIONS OF TESTICLE, &amp;c.

No.	Age.	Disease and Remarks.	Days in House.
1	53	Cancer. Extensive ulceration. Greater part of scrotum removed.	64
2	54	Recurrent cancer in scrotum. Testicle removed	37
3	30	Chimney sweeper's cancer. Skin of thigh and groin invaded.	25(?)
4	46	Cystic disease of testicle.	15
5	41	Sarcoma of testicle.	26

## EXCISIONS OF VARIOUS TUMOURS.

No.	Sex and Age.	Disease and Remarks.	Days in House.
1	F. 30	Lipoma of shoulder.	11
2	M. 57	Lipoma on back.	15
3	M. 51	Multiple lipomata on arm.	38
4	M. 52	Multiple lipomata on legs.	7
5	M. 57	Multiple lipomata on chest and abdomen.	31
6	M. 41	Lipoma on neck.	6
7	F. 30	Fibro-lipoma of thigh.	16
8	F. 72	Lipoma on shoulder.	33
9	M. 64	Lipoma on shoulder.	10
10	F. 51	Cystic tumour on neck.	19
11	M. 39	Fibro-cystic tumour in neck.	16
12	M. 28	Fibroma on buttock.	26
13	M. 50	Fibroma in calf of leg.	33
14	F. 30	Fibro-lipoma in thigh.	16
15	M. 12	Recurrent fibroid on leg.	22
16	M. 34	Fibroid tumours on arm. Recurred. Amputation.	121
17	F. 53	Scirrhus over sacrum. Wound suppurated.	78
18	M. 27	Submental glands from epithelioma of lip.	17
19	M. 69	Epithelioma on back of hand.	32
20	F. 64	Epithelioma of eyelids.	21
21	M. 69	Epithelioma of nose.	6
22	F. 59	Extensive epithelioma of vulva. Recurrence.	130
23	M. 35	Recurrent cancer of tongue.	37
24	F. 35	Cancer of tongue.	18

EXCISIONS OF VARIOUS TUMOURS—*continued.*

No.	Sex and Age.	Disease and Remarks.	Days in House.
25	M. 38	Epithelioma of penis. Amputation and recurrence in anus. Removal from anus, and secondary recurrence in eight months.	128
26	F. 21	Enchondroma on thumb.	3
27	F. 23	Enchondroma of metatarsus.	29
28	F. 18	Osteo-enchondroma of great toe.	7
29	M. 8	Fibro-enchondroma on nose.	22
30	F. 55	Epulis of lower jaw.	1
31	F. 56	Naso-pharyngeal growth. Ecraseur.	3
32	M. 35	Large fibroid polypus of rectum. Ecraseur.	49
33	F. 16	Large exostosis of shaft of femur.	
34	M. 27	Chronically inflamed bursa patellæ.	8
35	F. 25	Suppurating bursa patellæ.	19
36	F. 33	Cartilaginous bursa patellæ.	20
37	F. 10	Exostosis on tibia.	9
38	M. 13	Nævoid tumour on chest.	29
39	M. 11	Cystic tumour on temple.	7
40	F. 45	Polypus of uterus. Ecraseur.	16
41	F. 40	Intra-uterine pedunculated growth. Ecraseur.	18

In the foregoing table small tumours, such as small nævi, epithelioma of lips or eyelids, sebaceous tumours of the scalp, &c., are excluded.

60 removals of tumours of all sorts are recorded; 34 in females, of which 14 were excisions of the breast; 26 in males, of which 5 were removals of testicle.

No death resulted.

The average stay in the house after excisions of the breast was 27 days. The shortest periods were 7, 8 and 12 days. In the other cases, the individuals are too variable for the average to be of any value.



## TABLE OF COMPOUND FRACTURES.

No.	Sex and Age.	Nature of Injury and Remarks.	No. of Days in House.
1	M. 41	Compound comminuted fract. of tibia, simple fr. of fibula. Barrel of beer fell on it.	82
2	F. 74	Compound fr. of ulna, simple fr. of radius. Fall down-stairs.	11
3	M. 20	Compound fr. of clavicle, laceration of lungs, emphysema. Laceration of scalp.	44
4	M. 20	Compound fr. of tibia and fibula; much bruising and laceration. Fr. of humerus and clavicle.	103
5	M. 43	Compound, very oblique fr. of tibia; protruding fragment removed. Fibula comminuted. Delayed union.	143
6	M. 40	Compound fr. of tibia, simple fr. of fibula. Cutaneous laceration.	30
7	M. 19	Compound fr. of tibia, fibula comminuted.	26
8	M. 33	Compound fr. of tibia, fr. of fibula. Bruising of skin.	57
9	F. 66	Compound fr. of tibia, slight comminution; fr. of fibula.	73
10	M. 5	Tibia and fibula much comminuted and exposed along mid. $\frac{3}{4}$ . Extensive laceration of tissues. Run over.	102
11	M. 35	Compound fr. of right humerus. Fr. of left radius exposing wrist joint.	147
12	M. 45	Compound fr. of tibia, slight comminution. Fibula fractured. Much bruising and laceration.	82
13	M. 53	Compound comminuted fr. of tibia; fr. of fibula. Emphysema.	45
14	M. 40	Compound fr. of tibia; fibula entire.	44
15	M. 26	Compound fr. of tibia, much comminution, fragments removed. Fr. of fibula. Free hæmorrhage. Run over.	61
16	M. 13	Compound fr. of tibia into ankle joint; fr. of fibula. Much laceration. Fall of stones in quarry.	39
17	M. 46	Crush of left foot and ankle; amputation of leg. Compound comminuted fr. of right tibia and fibula. Much laceration. Fragments of bone removed. Delayed union.	167
18	31	Compound comminuted fr. of tibia into ankle joint; fr. of fibula. Delirium tremens. Pleurisy.	113

TABLE OF COMPOUND FRACTURES—*continued.*

No.	Sex and Age.	Nature of Injury and Remarks.	No. of Days in House.
19	F. 27	Compound comminuted fr. of tibia near ankle; fr. of fibula. Much effusion into the ankle joint.	41
20	M. 14	Compound fr. of tibia; fr. of fibula. Free bleeding.	30
21	M. 52	Great comminution of tibia, large contused and lacerated wound; fibula fractured. General bruising.	77
22	M. 78	Compound fracture of tibia; fr. of fibula. Much contusion. Wound extensive.	40
23	M. 10	Crushed elbow joint; cavity freely exposed. Extensive lacerated and contused wounds. Run over. Moveable joint.	58
24	F. 8	Compound fr. of tibia; fibula entire. Hæmorrhage.	36
25	F. 70	Compound comminuted fr. of ulna, compound fr. of radius.	17
26	M. 76	Compound fr. of tibia; compound comminuted fr. of fibula near ankle joint. Great contusion.	37

In the above table only those cases which might be classed as dangerous to life or limb are noted. Compound fractures of the fingers or toes, crushed hands (which were very numerous) and crushed feet are excluded. Compound fractures of the skull are referred to elsewhere. In two cases, one of crushed leg, the other of crushed hand and forearm, attempts to save the limb were unsuccessful; in all the other cases, twenty-six of the most severe of which appear in the above table, recovery took place with bony union and useful limbs. The plan of treatment was the same in all—that of Professor Lister. Few points call for special notice. Four patients were over 70 years of age. In three of the cases the fracture was into a joint cavity, and a moveable joint resulted in all. In two cases the gradual absorption of sloughed tissue, which remained free from odour, was observed.

## OPERATIONS FOR NECROSIS AND CARIES.

No.	Sex and Age.	Nature of Disease and Operation.	No. of Days in House.	Result.
BONES OF SKULL.				
1	F. 37	Caries of mastoid cells. Gouging and Scraping	7	Cured.
2	F. 27	Necrosis of hard palate. Sequestrotomy. Opening closed by plastic operation afterwards.	89	Cured.
3	F. 8.	Caries of malar bone. Sinus in cheek. Gouging and scraping.	9	Cured.
4	M. 3.	Caries of malar bone. Sinus in lower eyelid. Scraping through sinus inside mouth.	79	Cured.
5	M. 32	Necrosis of angle of lower jaw. Sequestrotomy. Scraping sinus and chloride of zinc.	45	Cured.
6	F. 11	Necrosis of ascending ramus of lower jaw. Sequestrotomy.	51	Cured.
7	F. 24	Necrosis of horizontal ramus of lower jaw. Suppurative periostitis. Sequestrotomy.	44	Cured.
8	M. 52	Necrosis of comminuted portion of fractured lower jaw. Sequestrotomy.	43	Cured.
BONES OF UPPER LIMB.				
9	F. 29	Necrosis and caries of scapula, humerus and clavicle. Sequestrotomy and scraping. Excision of joint afterwards.	136	Excision.
10	M. 13	Caries of shoulder joint. Scraping off carious bone.	3	Relief.
11	M. 31	Caries of head of humerus and scapula. Gouging, scraping, and chloride of zinc.	85	Improved.
12	M. 22	Extensive necrosis of shaft of humerus. Removal of several large sequestra.	Progressing favourably.	
13	M. 23	Necrosis of head and shaft of humerus. Removal of several sequestra.	Progressing favourably.	
14	M. 19	Caries of lower end of humerus. Gouging, scraping, and strong carbolic lotion.	35	Cure.
15	M. 20	Necrosis of the upper third of shaft of humerus. Gouging and sequestrotomy.	83	Improved.



OPERATIONS FOR NECROSIS AND CARIES—*continued.*

No.	Sex and Age.	Nature of Disease and Operation.	No. of Days in House.	Result.
BONES OF UPPER LIMB— <i>continued.</i>				
16	F. 12	Caries at elbow joint after excision. Scraping.	90	Cured.
17	M. 3	Necrosis of olecranon ulnæ. Sequestrotomy.	19	Cured.
18	M. 14	Extensive necrosis of shaft of humerus. Removal of large sequestrum by gouging.	67	Cured.
BONES OF PELVIS.				
19	M. 36	Sacro-Iliac disease. Removal of portion of ilium. Scraping and chloride of zinc.	62	Cured.
20	F. 14	Necrosis of crest of ilium. Removal of sequestrum. Gouging of carious portions.	120	Cured.
21	F. 44	Necrosis and caries of rami of ischium and pubes. Removal of necrosed, and scraping of carious, portions.	70	Cured.
22	F. 11	Sacro-Iliac disease. Gouging carious bone. Scraping and syringing of sinuses.	Improving.	
23	F. 12	Caries of crest of ilium. Gouging and scraping.	Improving.	
24	F. 45	Caries of symphysis pubis. Removal of necrosed and scraping of carious bone.	23	Cured.
25	F. 11	Caries of ilium near acetabulum. Morbus coxæ. Scraping of bone and sinuses.	46	Cured.
26	M. 36	Necrosis of ilium and caries of femur after excision of hip. Sequestrotomy and Gouging.	91	Cured.
27	F. 33	Caries of ilium and femur. Old hip disease with dislocation. Scraping bone and sinuses.	76	Cured.
FEMUR.				
28	F. 27	Caries of femur. Old hip disease and ankylosis. Scraping bone and sinuses, and chloride of zinc.	56	Relieved.
29	M. 15	Necrosis of shaft of femur. Sequestrotomy and scraping of sinuses.	148	Cured.

OPERATIONS FOR NECROSIS AND CARIES—*continued.*

No.	Sex and Age.	Nature of Disease and Operation.	No. of Days in House.	Result.
FEMUR— <i>continued.</i>				
30	F. 16	Caries of femur after excision of knee. Gouging, scraping and chloride of zinc.	88	Cured.
31	M. 15	Caries of internal condyle of femur. Gouging, scraping and chloride of zinc.	76	Cured.
32	M. 14	Necrosis of middle third of shaft of femur. Sequestrotomy.	30	Cured.
33	F. 16	Necrosis of lower third of shaft of femur. Gouging. Sequestrotomy.	Improving.	
BONES OF LEG AND FOOT.				
34	M. 23	Caries of lower end of tibia. Gouging and scraping.	77	Cured.
35	M. 15	Caries of upper end of tibia. Gouging and scraping.	87	Cured.
36	M. 15	Necrosis of shaft of tibia. Sequestrotomy.	231	Cured.
37	F. 6	Caries of tibia. Pulpy synovitis of ankle. Gouging and scraping of bone. Ankle opened.	370	Cured.
38	M. 10	Caries of os calcis. Gouging and scraping. Organised blood clot.	34	Cured.
39	F. 15	Caries in femur after excision of knee. Gouging and chloride of zinc.	211	Cured.
40	F. 18	Necrosis of shaft of tibia. Sequestrotomy.	223	Cured.
41	M. 14	Necrosis of shaft of tibia. Sequestrotomy.	88	Cured.
42	F. 14	Necrosis of shaft of tibia. Sequestrotomy.	40	Cured.
43	M. 15	Necrosis of mid. $\frac{1}{3}$ of shaft of tibia. Caries near ankle. Sequestrotomy and scraping.	97	Cured.
44	F. 9	Caries of lower end of tibia and astragalus. Partial bony ankylosis. Gouging and scraping.	231	Cured.
45	F. 18	Caries and necrosis of head of tibia. Gouging, removal of sequestrum, and scraping.	77	Cured.
46	M. 13	Caries of os calcis. Gouging and scraping.	90	Cured.

OPERATIONS FOR NECROSIS AND CARIES—*continued.*

No.	Sex and Age.	Nature of Disease and Operation.	No. of Days in House.	Result.
BONES OF LEG AND FOOT— <i>continued.</i>				
47	M. 13	Necrosis of shaft of tibia. Sequestrotomy.	29	Cured.
48	F. 10	Caries of lower end of tibia. Gouging and scraping.	146	Cured.
49	M. 18	Necrosis of mid. $\frac{1}{3}$ of shaft of tibia. Sequestrotomy.	18	Cured.
50	M. 17	Necrosis of shaft of tibia. Sequestrotomy.		Improving.
51	M. 40	Caries of tibia near ankle joint. Gouging, scraping, and chloride of zinc.	66	Cured.
52	F. 12	Disease of calcaneum and astragalus. Gouging and scraping. Organised blood clot.	25	Cured.
53	F. 19	Superficial necrosis of tibia. Strumous abscesses in intermuscular fascia. Scraping.		Improving.

The operations for removal of carious bone by the gouge or by Volkmann's spoon have been attended with very satisfactory results. In many cases it was possible, even though old sinuses existed, by the use of chloride of zinc or carbolic acid, to render the wounds aseptic and treat them according to Lister's method. In these cases the healing and closure was more rapid, there was less suppuration, and, it may be said, no constitutional disturbance. In two cases, cavities left after gouging out a carious os calcis were filled with organised blood clot, and in these the cure was very rapid.

In cases of necrosis, the sequestrum in many instances had not become loose when it was removed. It is doubtful whether the increased facility of operation when the dead bone becomes separated, compensates for the weakening influence of suppuration and the deformity from prolonged deposit of new bone which result from delay in operative interference.

No death resulted from operation in the 53 cases.



## SHORT NOTES OF INTERESTING SURGICAL CASES.\*

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### CASE I.

*Compound depressed fracture of skull. Lacerated wound of longitudinal sinus. Trephining. Recovery.*

UNDER MR. TIBBITS.

S. M., æt. 24, shoemaker. 65 days.

Patient was admitted in a state of insensibility, with numerous contused and lacerated wounds of the scalp, and an extensive compound depressed fracture on the vertex of the skull. The injuries had been caused by blows from a fire-tongs. On removing some of the bony fragments, it was found that a large spicule had entered the longitudinal sinus, and the slightest movement of the bone caused an alarming gush of blood. After the use of the trephine and saw the surrounding depressed portions were elevated or removed, and the piece which perforated the sinus was left to the last. By means of a couple of tenacula and a torsion forceps, the wound in the sinus was closed instantaneously by an assistant when the spicule was removed, and very little blood was lost. The rent, which was about  $\frac{3}{4}$  in. long, was carefully stitched

\* Three of the cases occurred prior to 1878. The notes are taken from the ward case-books ; but, in almost every instance, the surgeons have seen and corrected the manuscript.—J. G. S.

up with catgut sutures. The scalp was shaved, the wounds were thoroughly washed with strong carbolic lotion, brought together with horsehair sutures, and treated antiseptically. The patient made an excellent recovery without the appearance of a bad symptom. The bone had been removed over an area of nearly four square inches, and when the patient was last seen, six months after, very little new bone had developed.

#### CASE II.

*Severe compound depressed fracture of skull. Extensive laceration of scalp. Trephining. Recovery.*

UNDER MR. GREIG SMITH.

A. B., æt. 43, mason's labourer. 29 days.

Patient fell from a height of twenty feet, striking his body and head on various objects in the descent. The fracture was caused by an upright bar of iron impinging on the skull midway between the ear and the occipital protuberance. The overlying tissues were much torn and bruised, and a large area of his scalp was lacerated and lay in shreds on the peeled cranium. There were also extensive bruises over chest, shoulders, and abdomen. Patient alternately comatose and convulsed. The depression, which was very deep, extended over a surface of about four square inches, occupying the posterior inferior angle of the right parietal bone, and contiguous portions of the occipital and temporal bones. It evidently pressed on the lateral sinus. After the use of the trephine and Hey's saw the depressed portions were elevated, and a loose piece of bone, about an inch square, was removed. The wounds were carefully washed with carbolic lotion warmed, brought together over drainage

tubes with horsehair sutures, and dressed with boracic lint. All of the wounds, except that over the fracture, were healed on the fourth day without the appearance of a drop of pus. Pus continued to discharge from the cavity over the fracture for three weeks, and at the end of that time he was discharged well. No cerebral symptom appeared during the treatment.

### CASE III.

*Compound depressed fracture of skull. Removal of fragments.  
Recovery.*

#### UNDER MR. STEELE.

B. W., æt. 15, office boy. 55 days.

The injury was inflicted by a blow from a stone about a pound in weight. The patient was only slightly stunned by the blow, and walked, unaccompanied and unassisted, more than a mile to the Infirmary, appearing in the casualty room in a perfectly conscious and collected condition. At the top of the forehead was a large jagged and contused wound overlying an extensive comminuted and depressed fracture of the frontal bone. Six fragments varying in size from a shilling to a threepenny piece were removed, and the rest of the fractured portions were elevated and left in position. The wounds were washed with strong carbolic lotion, and treated antiseptically. On the third day the temperature was normal, and continued so during treatment. A steady and uninterrupted recovery was made; no brain symptom appeared, and the patient took food, and read, and talked, almost as if he had been in perfect health throughout. He was discharged with the brain visibly pulsating under the cicatrix, and went immediately to work.



## CASE IV.

*Acute necrosis and osteomyelitis of Femur. Pyæmia. Amputation. Albuminuria, pleurisy, peritonitis. Secondary amputation. Recovery.*

UNDER MR. TIBBITS.

J. F., æt. 17, carter. 109 days.

A fortnight prior to admission patient received a severe blow on the lower part of the left thigh. The same evening there was great pain at the seat of injury; the patient felt very ill, and had a severe rigor followed by profuse sweating. The pain increased and became throbbing in character, and the skin over the lower half of the thigh became hot, red, and tense. Four days after the infliction of the blow pain, redness, and swelling appeared in the right shoulder and the right knee; and two days later the same symptoms manifested themselves in the hip and first metacarpo-phalangeal joint, also of the right side. He continued in this condition, having occasional rigors, and emaciating rapidly up till admission. A deep abscess was detected above the left knee joint (at the seat of injury), and laid freely open down to the bone. About four inches of the lower end of the femur was found denuded of periosteum. A fortnight afterwards, the patient having lost ground, and the purulent discharge becoming more profuse, the thigh was amputated through the middle third. The medullary cavity in its lower half was found to be filled with foetid pus, the epiphysis was completely separated from the shaft, and the whole femur in its lower half was denuded of periosteum. Two days after operation symptoms of acute nephritis, with albuminuria, renal casts, &c., set in, and lasted for three weeks. Four weeks after this he had a sharp attack of right pleurisy, of which

he slowly recovered. Symptoms of peritonitis now set in; but they soon subsided. The stump, which had healed kindly, became red and painful two months after operation; an abscess formed, and several pieces of necrosed bone were removed. Ten weeks after admission the boy was sent to a convalescent home, the stump having healed, and the symptoms of abscess formation in the joints having disappeared. A month subsequently he was readmitted with periostitis and thickening, accompanied with great pain, in the end of the stump. Palliative treatment proving ineffectual, secondary amputation was performed just below the trochanter minor. An excellent recovery took place, and the patient was sent home cured.

#### CASE V.

*Acute necrosis of tibia. Pyæmia. Amputation. Recovery.*

UNDER MR. TIBBITS.

I. D., æt. 8, schoolboy. 129 days.

A fortnight before admission patient was kicked on the shin by a schoolfellow. Next day the limb was swollen and very painful, and the boy was confined to bed. On admission, the presence of pus could not be detected; but the pain, tension of skin, high temperature ( $104^{\circ}$ ) and profound constitutional disturbance, pointing to suppurative periostitis, two long incisions were made on the tibia, and some pus escaped. His condition, however, got worse; he had several severe rigors, once with a temperature of  $105.8$ , and frequently with temperatures over  $104^{\circ}$ ; appetite for food failed, and he became much emaciated. Accordingly, thirteen days after admission, amputation was performed through the knee-joint. Part

of the flaps sloughed, and the condylar epiphysis necrosed. The pyæmic symptoms abated, and the patient made a slow recovery. No secondary abscesses or other complications appeared during the progress of the case.

#### CASE VI.

*Acute necrosis of femur, with arthritis of knee. Antiseptic incisions. Removal of sequestrum. Recovery.*

UNDER MR. CROSS.

W. M., æt. 13.

Admitted December 16th, 1878. Nine weeks prior to admission had been kneeling on the damp ground, and noticed a pain in his knee. In a day or two severe arthritis set in, and he was confined to his bed. When admitted to the Infirmary he complained of pain on moving the knee. It was swollen and subacutely inflamed; a small sinus existed on the inner side of the joint, which emitted scarcely any discharge. No history of sudden relief to the joint pain or of discharge at the sinus could be ascertained.

For a fortnight the boy lay without any constitutional disturbance. The thigh was slightly boggy, but not tender, and the knee did not seem inflamed, though it was enlarged. His temperature did not reach 100°.

On the night of December 30th temperature reached 102; pulse, 148; great pain complained of at the lower end of the femur. Next day a long incision was made behind the adductor magnus tendon from the sinus downwards; the popliteal surface of the femur found quite bare. Three long incisions were carried down to the bone on the outside of the thigh, and the whole shaft was found to be bare from the trochanter to the external



condyle. The wounds were syringed out with 40 grains to the ounce of zinc chloride, drained, and dressed antiseptically. The knee joint was not interfered with. A Gooch splint was applied; the limb was dressed daily for three days, every other day for ten days longer, and then required dressing every four days. For a week after the operation the highest temperature recorded was 101; during the next three weeks, 100; it then became normal.

On April 16th a tendency of the knee to bow outwards was opposed by applying a side splint.

July 18th. A sequestrum, two inches in length, the whole thickness of the diaphysis of the femur, was extracted through an incision along the biceps tendon, a large quantity of new bone requiring removal from its surface by the chisel and hammer. Six weeks after this operation he returned home with a stiff knee and sound lower extremity, a small sinus remaining at the site of operation.

On Jan. 8th, by enlarging this sinus, a piece of dead bone, more than two inches in length, and lying superficial, was taken out, together with several smaller pieces. He has been discharged from the hospital with a mere superficial healing wound.

#### CASE VII.

*Acute necrosis of head of tibia. Disorganisation of knee joint. Pyæmia. Albuminuria. Death.*

UNDER MR. BOARD.

J. M., æt. 20, collier. Four days.

Patient's right knee had been weak and occasionally painful during the previous twelve months, but he had always been able to walk about and work. Six days

before admission, without apparent cause, he felt severe pain in that knee, and it suddenly became much swollen. On admission the patient was in a condition of low delirium, with brown dry tongue, flickering pulse and sub-sultus tendinum, and was troubled with a frequent hacking cough. The right knee joint presented all the signs of acute suppuration, and there was evidently a large abscess over the inner tuberosity of the tibia. The knee joint was freely laid open on both sides, and the abscess over the tibia was also evacuated. About a pint of unhealthy sanious pus escaped. The inner tuberosity of the tibia was denuded of periosteum. T., 105; p., 142; r., 37. Next day it was noticed that he had almost complete left hemiplegia, and his urine was found to be highly albuminous. Amputation, under these circumstances, was deemed hopeless, and the patient gradually sunk and died. There was free perspiration, but no shivering; sickness and vomiting was a prominent symptom.

## CASE VIII.

*Pyæmia after labour. Acute suppuration of knee-joint.*

*Amputation of thigh. Recovery.*

UNDER MR. BOARD.

S. R., æt. 46, married woman. 61 days.

Patient's labour had not been difficult, though there appeared to have been much hæmorrhage, and a good recovery was made. A month after confinement she woke up one night with excruciating pain in the knee joint, and found her leg and foot much swollen. Next day a medical man, who was called in, ordered the application of poultices and fomentations, and six weeks after-

wards opened a large abscess in the calf. During this time the patient had lost much flesh, and suffered from night sweating and occasional rigors. The knee joint continued to swell, and on admission was found full of pus and completely disorganised, and the tibia dislocated backwards and rotated outwards. She was suffering from an attack of acute bronchitis on admission. Amputation through the lower third of the thigh was performed, and the patient made a slow recovery without the appearance of any further symptoms.

#### CASE IX.

*Chronic disease of inner half of knee joint, with ostitis.  
Removal of diseased structures. Recovery with a movable joint.*

UNDER MR. CROSS.

O. H., æt. 11, schoolboy.

Patient had for two years and a half suffered from chronic disease on the inner side of the knee joint. In April, 1879, he was admitted into the wards with an ulcer, which did not apparently communicate with the knee joint over the inner side of the knee. The inner condyle and tuberosity were enlarged and the knee scarcely movable, but he was able to walk on it and no deep mischief was present. The bony points on the outside of the joint were normal, but there was some thickening of the capsule.

He was kept in bed, the leg on a straight splint and the ulcer healing at its edges, but on April 26 he complained of being feverish. The next evening his temperature reached 103·5. He had no appetite, and complained of pain in the knee, which was swollen and tender under the ulcer. April 28th, no improvement.



The ulcer was well swabbed with 40 grain solution of zinc chloride, the limb washed with strong carbolic lotion, and an incision made into the joint. The internal fibro-cartilage was absent, and the inner condyle and internal tuberosity were hollowed out, forming a kind of cyst, which was filled with granulation tissue. Volkmann's spoon and the gouge were freely used, the cyst emptied, and a considerable quantity of carious bone removed from the condyle and tibia and from the inner facet of the patella.

The knee could now be freely moved, the outer half of the joint not appearing diseased. A horsehair drain was passed through behind the external lateral ligament from the site of operation, which was well washed out with strong carbolic lotion and dressed on Lister's method. Next day the temperature had fallen two degrees, and no unfavourable symptom occurred until May 3rd. He then complained of great pain inside the knee above the condyle. An incision here down to the bone freed a small amount of pus; a drainage tube was introduced through the wound. Next day the temperature had fallen to 100. On the 7th a little pus was escaping along the horsehair drain. The other two wounds were filling, and the tubes required shortening. On the 12th the horsehair drain was drawn down, so that its upper end should be in the cavity of the joint. Drain removed from femoral incision. Two days after the horsehair drain was drawn out. For a day or two the outer side of the joint was distended with fluid, but the patient felt no inconvenience. After May 22nd, 28 days from operation, the joint required dressing only once a week. The outer side was still containing fluid. This gradually diminished, and he was discharged on August

14th, walking well upon the knee, which could be flexed about 60° from extension. In January, 1880, the boy was seen pushing a hand-cart, and possessing considerable freedom of movement of the knee joint.

CASE X.

*Acute necrosis of tibia. Free suppuration for three months. Disorganisation of ankle joint. Amputation. Recovery.*

UNDER MR. PRICHARD.

R. R., æt. 15, housemaid. 151 days.

For a fortnight prior to admission patient had suffered intense and deep-seated pain all over the right leg, with profound constitutional disturbance, sleeplessness and occasional rigors. When seen the leg was swollen, the skin tense, red, hot and tender, but the presence of pus could not be detected by palpation. There was fluid in the knee and ankle joints. Two long incisions were made over the internal surface of the tibia down to the bone, and about six ounces of pus escaped. The openings were made with antiseptic precautions, but the subsequent course was not aseptic. Large quantities of healthy pus were discharged daily, and after three months' waiting it was evident that she was losing ground and beginning to suffer from hectic. The condition of the limb having been examined carefully while the patient was under chloroform, it was thought that amputation offered the best chances of recovery. Carden's operation was performed antiseptically, and primary healing took place. The patient was in the garden on the third day. She was discharged cured a month after operation.

No cause could be discovered, unless wet feet might be so reckoned.

CASE XI.

*Acute necrosis of tibia. Free incisions. Removal of dead bone. Recovery.*

UNDER MR. CROSS.

J. C., æt. 14, servant girl. 68 days.

Without assignable cause, patient suddenly felt a severe pain along the front of the left leg, and became so ill that she was obliged to take to bed. Next morning Mr. Omerod, of Westbury, saw her, and, diagnosing suppurative periostitis, made an incision, four inches in length, along the inner surface of the middle of the tibial shaft. Poultices were applied, and free suppuration set in. A fortnight afterwards she was sent to the Infirmary. A thin scale of bone four and a half inches long, and about an inch broad, was removed from the inner surface of the tibia; and, a month afterwards, another piece less than half the size came away of its own accord. An excellent recovery was made.

CASE XII.

*Acute necrosis of lower jaw. Intracranial suppuration. Death.*

UNDER MR. TIBBITS.

J. H., æt. 37, sugar baker. 14 days.

Patient's illness began six weeks before admission with pain and tenderness at the right angle of the jaw. No cause could be given. Three weeks before admission the side of the jaw, face, and neck having become greatly swollen, he had severe rigors followed by profuse sweating. About this time he became deaf in the right ear, and suffered from severe pain in the *left* orbit. Soon



afterwards the left eye was protruded, and the eyelids became puffy and œdematous. At the same time he complained of a severe lancinating pain in the top of his head. On admission a free incision was made over the angle and along the horizontal ramus of the jaw, and a large quantity of fœtid pus was evacuated. Nearly the whole of the right half of the jaw was denuded of periosteum. The base of the right lung was found to be consolidated. The pyæmic symptoms abated in acuteness; but the pneumonia did not clear up, and the patient gradually sank and died.

The autopsy revealed several pyæmic abscesses in the right lung, and a small encysted collection of pus in the left orbit. Pus was found to have entered the skull through the vaginal plate of the temporal bone, by the carotid canal and along the course of the cavernous sinus. The pus in the left orbit seemed to be in communication with a collection in the circular sinuses.

#### CASE XIII.

*Pyæmia from a cancerous sinus. Cellulitis of neck and face. Suppuration of knee joint. Recovery from pyæmic condition with movable joint.*

UNDER MR. CROSS.

G. P., æt. 27, sailor.

In September, 1878, the patient had a large epithelioma removed from the lower lip, and also an enlarged gland over the right cornu of the hyoid bone. On Nov. 28th, the growth having recurred, the right half of his lower lip and several enlarged glands were removed. Having been at sea for nine months, he returned with a fluctuating tumour in the right submaxillary region, and

at the angle of the jaw a solid tumour as large as a hen's egg. The abscess was opened antiseptically, and the discharged matter was found to contain epithelial elements. In three weeks he went home, the sinus still discharging, with orders to return for antiseptic dressing. A week later he re-appeared in a very critical condition, with diffuse cellulitis of the left side of the face and neck. The sinus was syringed out with a strong antiseptic lotion, and deep incisions were made in the neck. During the next fortnight he had frequent rigors with high temperatures (between  $101^{\circ}$  and  $105^{\circ}$ ), he perspired freely, his tongue was coated and dry, and at night he was delirious. Pulse 120-130; respirations 38-42. On the third week the left side of his face became paralysed, and the right ear deaf; and he continued to have rigors. On the sixth week his left knee joint was found to be full of pus, and was opened antiseptically. Ten days later an abscess appeared in the left sterno-clavicular joint, and a little afterwards another over the left sacro-iliac joint. During the next month, with the exception of several exacerbations, his condition gradually improved; and three months after it was opened he was able to walk about with a movable knee joint. He was dismissed in fair condition of health, but when last seen (three months subsequently) the epitheliomatous growth was still advancing.

## CASE XIV.

*Chimney sweepers' cancer involving skin of thigh and groin.  
Removal. Rapid recovery.*

UNDER MR. DOWSON.

G. F., æt. 30, chimney sweep. 39 days.

The disease appeared two years previously as a hard

grey wart on the anterior surface of the right side of the scrotum. Since then it had steadily increased in size, till, on admission, it had involved the whole of the right and part of the left side of the scrotum, and about an inch and a half of the contiguous skin of the thigh and groin. The patient was in such a feeble condition from pain, sleeplessness and prolonged suppuration, and the disease was so extensive, that an operation was deemed scarcely justifiable, and was undertaken only at the man's earnest request. The whole of the diseased and fungating mass was removed along with the right testicle. Sufficient skin was left to cover the left testicle, and the rest of the exposed surface was covered as much as possible by drawing together the surrounding integument. Carbolic oil and terebene and vaseline were used as dressings. The wounds healed up with great rapidity, and the patient quickly improved in health. On discharge, he was able to resume work at once. Up to date (one year afterwards) there has been no recurrence of the growth. The minute structure of the growth was epitheliomatous.

CASE XV.

*Extensive Epithelioma in an old ulcer of the leg.*

*Amputation. Recovery.*

UNDER MR. CROSS.

M. W., æt. 63, labourer.

Patient has had an ulcer on the inside of the middle of the right leg for twenty years. During the last three months the aspect of the wound had changed, its surface becoming raised, nodulated and sloughy, and its edges hard and much elevated. It had also become very painful. Amputation was performed by anterior skin flap, with



a short posterior flap cut by transfixion. The tibia was sawn two fingers' breadths below the tubercle. Lister's antiseptic method was strictly followed. The stump was dressed on the two days following the operation, on the eighth and on the thirteenth days. There was scarcely even serous discharge throughout. On the thirteenth day the stitches and drain were removed, and he was allowed to move about carefully in bed; on the nineteenth day only a superficial wound remained, and boracic lint was used instead of gauze. On the twenty-third day he was well, but the epithelium did not fully cover the scar until the thirty-second day—eight dressings in all. The structure of the growth was typically epitheliomatous. The periosteum underlying it was enormously thickened, and a layer of new bone, one-third of an inch in depth, was laid down over the old. Neither periosteum nor bone were invaded by the malignant growth.

*Note.*—A precisely similar case was admitted under Mr. Greig Smith, but permission to amputate was not granted.

#### CASE XVI.

*Large epithelioma in cicatrix of Chopart's amputation of the foot. Amputation of the leg. Recovery.*

UNDER MR. GREIG SMITH.

J. W., æt. 46, commercial traveller. 24 days.

Twenty-eight years ago patient's left foot was crushed by being run over, and Chopart's amputation was performed. The wound never thoroughly healed, but he was able to walk about with it. For six months prior to admission the granulating surface had been growing larger in area, and had been getting hard and nodular. On admission the whole end of the stump was covered

by typically epitheliomatous structure, and the tissues of the heel and ankle were involved to a slight extent. The limb was slightly œdematous and much wasted. The patient was very weak, had evening temperatures of over 100°, and suffered from night sweating. Amputation by posterior flap was performed at the junction of the upper and middle thirds of the leg. The wound healed by first intention under two antiseptic dressings. The os calcis was found to be carious in its interior.

#### CASE XVII.

*Extensive epithelioma of lower lip and chin. Cheiloplasty.*

UNDER MR. GREIG SMITH.

F. H., æt. 77, labourer. 33 days.

The disease, which had been growing for more than a year, involved the whole of the skin of the lower lip and most of that of the chin. On the right side it extended beyond the commissure of the lips and invaded the tissues of the cheek. There was no glandular invasion. One long straight incision was carried from the cheek beyond the right angle of the mouth down to the hyoid bone. Another incision from the left corner of the mouth met the first one a little below the lower margin of the jaw. A rectangular flap, raised from the left side of the chin and neck, was made to form the under lip and cover the upper portion of the exposed surface. A little dissection in the hyoid region provided a covering for the rest. There was abundant vascular supply, and the cut vessels were closed by forcipressure (Dieffenbach's). The incisions all healed by first intention; there was slight suppuration on the inner surface of the lower lip for a few days. Boracic acid lotion and dress-

ing were employed. The facial appearance was hardly changed.

## CASE XVIII.

*Popliteal aneurism cured by digital compression. Cellulitis of leg nine months afterwards. Recovery.*

UNDER MR. BOARD.

J. O., æt. 36, brewer. 62 days.

Nine months ago patient was admitted with left popliteal aneurism as large as a closed fist. Treatment by flexion was tried and failed. Digital compression on the femoral for four hours was followed by consolidation of the sac and cure. Just before the compression was removed he had an attack of shivering. After pursuing his ordinary laborious avocation for nine months, he returned in a critical condition from extensive cellulitis of the left leg and thigh. No cause could be elicited; it had been gradually coming on for a week. Free incisions were made, and more than two pints of pus were evacuated. Under antiseptic treatment a rapid recovery was made, and the patient went out walking with slight lameness. The leg is now (three months afterwards) quite well, though there remains some fulness in the popliteal space.

## CASE XIX.

*Psoas abscess. Antiseptic opening. Speedy recovery.*

UNDER MR. PRICHARD.

Annie C., æt. 10. 35 days.

Angular curvature of the last two dorsal and first lumbar vertebræ had existed for three years. On admission a large abscess bulged on the inner side of the right thigh, giving free fluctuation above and below Poupart's



ligament. A free opening was made into the sac, and a large quantity of curdy pus escaped. For five days after the operation the child had occasional rigors and vomiting, with high temperatures; but on the sixth day the temperature was normal. For the first week the dressings were changed daily on account of the free discharge. At the end of a fortnight only serum escaped, and in three weeks the gauze dressings were left off. She had gained flesh and strength considerably; and a month after the operation a Sayre's jacket was put on, and she was permitted to get up and walk about. Very slight serous oozing, just enough to soil the dressing, continued for two months. The plaster of Paris jacket was kept on for six months, and when it was removed the child seemed quite well.

#### CASE XX.

*Psoas abscess. Antiseptic opening. Rapid recovery.*

UNDER MR. PRICHARD.

M. R., æt. 28, married woman. 28 days.

Patient's back had been weak for six months, and a swelling had been growing on the inside of the right thigh for a month. There was angular curvature, with tenderness on pressure on the spines of the last dorsal and first two lumbar vertebrae. There was no pain in the recumbent posture. A large fluctuating tumour existed below Poupart's ligament, the right thigh at the level of the great trochanter measuring 21 inches in girth, the left 15 inches. An opening was made in the ordinary way near the apex of Scarpa's triangle, and a drainage tube inserted. The wound was dressed four times, and on the seventeenth day the incision had skinned over. There was no pyrexia, and the patient

rapidly gained strength. A Sayre's jacket was put on, and a month after operation she was able to walk home a distance of two miles. She still (five months afterwards) continues in excellent health.

## CASE XXI.

*Large chronic abscess connected with lower ribs. High temperatures, hectic and wasting. Antiseptic opening. Rapid closing without suppuration.*

UNDER MR. GREIG SMITH.

C. D., æt. 11, schoolboy. 50 days.

Patient had been losing flesh for some months, and a month prior to admission first noticed a swelling in the left side, between the ribs and the crest of the ilium. On admission this tumour was as large as two closed fists and distinctly fluctuated. The boy had lost his appetite for food; his evening temperatures reached  $103^{\circ}$ ; and he was wasted almost to a skeleton. An opening was made, and 26 ounces of curdy pus were evacuated. No diseased bone could be discovered. The temperature next day was normal, and continued so till the end, and constitutional improvement rapidly set in. Free discharge, remnants of the original collection, took place for a few days; thereafter, with four more antiseptic dressings at intervals of a week, the cavity had closed. He was kept in the house for three weeks afterwards, to make sure that no residual collection formed.

*Note.*—The three preceding cases are recorded as typical of the behaviour of chronic abscesses opened after Lister's method. After the proper contents of the abscess have been evacuated there is a slight discharge of sero-pus or serum for a few weeks, and the walls of the abscess cavity unite without suppuration. The temperature quickly becomes normal; and, if the treatment runs an aseptic course, continues so till the end. Constitutional disturbance ought to be nil. Many similar abscesses, from disease of the spine, pelvis, hip, &c., treated in like manner have had like results.—J. G. S.

## CASE XXII.

*Curious disintegration of Psoas muscle. Wasting. Death.*

UNDER MR. DOWSON.

S. R., æt. 26, labourer. 53 days.

The patient was admitted for ulceration of the rectum, and was treated with the local application of chromic acid, and milk diet. As the condition of his rectum improved, he gradually got weaker with foul tongue, fœtid breath, great drowsiness, and occasional delirium. There was no pyrexia. Repeated examinations in the pelvis and elsewhere threw no light on the cause of his sinking. He had no pain anywhere.

At the *post mortem* examination the substance of the right psoas muscle was found to be replaced by a dark brown fœtid fluid composed of blood, mingled with debris of muscular tissue. The fascia around the psoas was neither distended nor collapsed, but had, as nearly as possible, the same appearance and dimensions as that over the left muscle, which was perfectly healthy. Only a few fibres of the upper portion of the muscle were left attached to the fascia; the contents of the sheath were simply the grumous fluid described. The most careful dissection revealed no evidence of aneurism, or bony disease in femur, pelvis or spine. His organs were all healthy. The body was excessively emaciated.

## CASE XXIII.

*Genu valgum. Chronic simple synovitis, partial fibrous ankylosis. Osteotomy. Cure with movable joint.*

UNDER MR. GREIG SMITH.

H. D., æt. 24, baker. 62 days.

Patient has had knock knee of the right limb from



childhood. Two years before admission his limb was squeezed and the joint wrenched between two horses. He was then confined to bed for a week from synovitis of the right knee. Since then the joint has never been strong, he has continued to be lame, and has been frequently confined to bed for days at a time with attacks of inflammation in the knee. For one of these attacks he applied for admission to the Infirmary. The joint was subacutely inflamed, painful on pressure and movement; and was stiff in the semi-flexed position from old adhesions. Patella was immovable. A wedge osteotomy was done through the internal condyle, and the joint cavity was entered. The limb could not be perfectly straightened, apparently because of the external lateral ligament. When the incision had healed (three weeks afterwards) the patient was again put under chloroform with the view of dividing the external lateral ligament, and moving the joint freely. On the application of some force the external lateral ligament gave way with a snap, and the limb came perfectly straight. Free passive movement was applied six weeks afterwards, the patient having been walking in a stiff bandage the while. All treatment was then stopped, and the patient recovered with a straight, movable, and in every way normal limb.

## CASE XXIV.

*Genu valgum. Complete dislocation outwards of patella.  
Osteotomy. Cure.*

UNDER MR. GREIG SMITH.

S. G., æt. 17, shoemaker. 36 days.

Patient has had genu valgum of left limb since infancy. Three years ago he had a fall which, according to his

own account, caused dislocation outwards of the patella. Since then the patella had developed a tendency to slip off the trochlear surface on flexion of the limb; and when admitted, before the leg was bent to a right angle with the thigh, the patella had slipped completely on to the outside of the outer condyle, its inner and outer borders pointing directly forwards and backwards. When the limb was so flexed he could not extend it without the assistance of his hand either to replace the patella or push forward the leg. He walked very lame. A wedge osteotomy was performed through the inner condyle, the point of the wedge being in the intercondyloid space. The limb was easily straightened; and complete healing took place in a fortnight. Passive movement was instituted three weeks after operation, and he was walking without lameness in six weeks. There was no tendency to dislocation of the patella six months afterwards.

#### CASE XXV.

*Fracture of larynx. Subcutaneous emphysema.*

UNDER MR. DOWSON.

J. W., æt. 39, cabinet maker. 6 days.

The injury was caused by a blow across the throat with a poker. Both thyroid cartilages were found to have been fractured near the middle line. The fragments were freely movable on each other, and crepitus was readily elicited. There was considerable ecchymosis over the larynx, and the skin of the neck was puffed and swollen with infiltrated air. There was an irritating laryngeal cough with some expectoration and some pain in swallowing. Speaking was difficult, and his voice was

hoarse and husky. There was dyspnœa only when the cartilages were pressed upon. Patient made a good recovery.

#### CASE XXVI.

*Cystic tumour of larynx. Evacuation of contents by incision.  
Recovery.*

UNDER MR. GREIG SMITH.

P. A., æt. 27, labourer. 25 days.

Patient had been under treatment for the past two years at various institutions for gradual loss of voice and occasional dyspnœa, but with no good result. On admission he had absolute aphonia and some difficulty of breathing. Examination by means of the laryngoscope, revealed a large, smooth, rounded tumour, covered with pale mucous membrane, occupying the right half of the glottis, and completely overshadowing both vocal cords. On pushing up the larynx the tumour could be reached with the forefinger, and was found to have semifluid contents. Severe dyspnœa coming on a few days afterwards, the tumour was freely incised with a long knife carried through the mouth, while an assistant pushed upwards and fixed the larynx. About two drachms of pink gelatinous fluid were squirted out. Relief to breathing was immediate, but the voice did not at once recover itself. The tumour gradually shrivelled up, and the patient was on a fair way to recovery when he had to be dismissed for misbehaviour. Iodide of potassium was administered on account of an obscure history of syphilis.



## CASE XXVII.

*Large fibro-cystic tumour in carotid region of neck. Excision.  
Recovery.*

UNDER MR. LEONARD.

F. F., æt. 26, dressmaker. 10 days.

The tumour had been growing for two years, but had increased very little in size during the last six months. It was about the size of a closed fist, lay along the left side of the larynx and trachea, nearer the sternum than the lower jaw, and pushed apart the sterno-mastoid and trachea. There was but little pain in the tumour itself, no dyspnœa, and but slight dysphagia. There was considerable engorgement of the cutaneous veins. On excision the tumour was found to be firmly adherent to the underlying structures, particularly to the left ala of the thyroid cartilage. There was abundant venous hæmorrhage. The tumour was adherent to, but not structurally connected with, the thyroid gland. The wound healed by primary union, and the patient was discharged cured on the fourth day after operation. The tumour was not examined microscopically. It was fibro-cystic in nature. A calcareous plate was found in one of the cysts.

## CASE XXVIII.

*Enchondromatous tumour of parotid gland. Removal.  
Recovery.*

UNDER MR. STEELE.

C. B., æt. 35, domestic servant. 28 days.

The tumour had been growing for ten years in the right parotid region, and was about the size of a hen's

egg. It was well circumscribed, lobulated and hard, slightly adherent to the deep parts below, and freely movable above. There were shooting pains all over the right side of the head, and pricking, uneasy sensations in the tumour itself. On operation it was found that only the lower portion of the parotid gland was affected. There was some difficulty in dissecting it out. Microscopically the tumour was found to be composed of true cartilage, with patches of myxomatous transformation. The patient made an excellent recovery.

## CASE XXIX.

*Osteo-fibroma of thigh. Removal. Recovery.*

UNDER MR. PRICHARD.

C. L., æt. 30, tailoress. 20 days.

A hard rounded tumour, about the size of a hen's egg, loosely attached in the subcutaneous tissues at the back of the thigh, had been growing for nine months. The tumour lay in that part of the thigh which rested on the edge of a chair while she worked a sewing-machine. It was removed antiseptically, and the incisions healed by first intention.

Microscopically, the tumour was a curious mixture of fibrous tissue, fat cells, a few groups of typically formed giant cells, and some points of ossification. The fibrous tissue, which was well formed, wavy and sparsely nucleated, contained in its meshes many groups of fat cells and a general sprinkling of small, shrivelled, fat globules. The giant cells also occurred in groups, and were supported by a delicate fibrous network, in which lay also a good many round granular cells, similar to those found in sarcomata. The bony nodules were well formed, and

lay in the contiguity of the giant cells, which were evidently concerned in their formation.

CASE XXX.

*Excessive contortion of leg after compound fracture through knee joint. Bony ankylosis. Wedge osteotomy. Hæmorrhage from bone. Amputation. Recovery.*

UNDER MR. BOARD.

W. M., æt. 25, labourer. 46 days.

Eighteen years before admission the patient's limb had been crushed by the wheel of a heavily laden dray. The joint was freely laid open and the articulation completely destroyed. The deformity came on after he began to walk on the injured limb, and had gradually increased ever since. The bones of the leg were firmly ankylosed to the femur, and bent inwards to an angle of about 120°. The whole of the inner and the greater part of the outer tuberosity of the tibia had disappeared, and the shaft of the tibia seemed to be directly set into the posterior aspect of the end of the femur. The foot was much rotated inwards. The femur on the deformed side was an inch and a half longer than its fellow. A wedge osteotomy was performed on the outer and anterior aspect of the joint, and the limb was brought into fairly good position. Free sanguineous oozing, which could not be checked by the ordinary means, continued through the afternoon and night of the operation, and next morning the patient's condition had become so grave that it was decided to amputate through the lower fourth of the femur. The bleeding was from the whole of the cut surface of the end of the femur. A good recovery was made after amputation.



CASE XXXI.

*Dislocation forwards of the astragalus reduced by manipulation. Cure with movable joint.*

UNDER MR. DOWSON.

M. E., æt. 22, waitress. 18 days.

The accident was caused by a fall of nine feet from the top of steps on which she was standing. The sole of her foot violently struck the ground while the foot was extended, and she fell on her back. There was much swelling and bruising, with great tension of the skin over the dislocated bone. There was nothing particularly notable in the characters of the dislocation. The patient being profoundly under the influence of chloroform, the dislocated bone was replaced by pressing it backwards while the foot was forcibly drawn away from the leg. The limb was tightly bandaged to a side splint and laid flexed on a pillow. In three weeks the leg and foot were encased in an immovable apparatus, made by rubbing a saturated solution of gum and chalk into a worsted stocking, and the girl was made an out-patient. A complete cure resulted.

CASE XXXII.

*Fracture of calcaneum, drawing up of fragment by tendo Achillis, replacement and fixing in position of separated bone.*

UNDER MR. DOWSON.

T. C., æt. 60, gardener. 90 days.

The injury was caused by a fall from a height of twenty feet. His right heel struck violently against a stone; and, on attempting to get up, he felt something

snap and give way there. His foot was much bruised and greatly swollen. The protruding end of the calcaneum was fractured off, comminuted, and drawn two and a half inches up the leg. An incision was made over the seat of fracture, and the displaced fragment drawn downwards and fixed with silver wire in its normal position. Some portions of the comminuted fragments necrosed and came away, but the tendo Achillis adhered, and the patient recovered with a movable joint and a perfectly useful limb.

#### CASE XXXIII.

*Sacculation of female urethra. Operation. Cure.*

UNDER MR. GREIG SMITH.

E. B., æt. 34, married woman. 62 days.

The patient applied for admission with a large, rounded, boggy tumour, which filled up the whole of the vagina and protruded through the vulva. The growth of the tumour dated from ten years previously, when she was confined, after a tedious instrumental labour, of her sixth and last child. Coition had for six years been impracticable; and latterly there was great pain in the tumour, particularly on micturition and defœcation. Her condition had been much aggravated by wearing a pessary, which a medical man had applied apparently in the belief that she had cystocele. A careful examination shewed that the tumour had no connection with the bladder. Exploration of the floor of the urethra revealed a small opening which led into the cavity of the tumour. This opening was dilated, and the contents, a most offensive mixture of slimy pus and caseated matter, were evacuated. The thick walls of the cavity were freely swabbed with a

strong perchloride of iron solution, in hopes of setting up free suppuration. After suppuration had set in, an inflation ball pessary was kept in the vagina so as to keep the granulations in apposition. The swelling gradually diminished, and she ultimately got quite well.

The origin of the tumour had probably been an abscess in the areolar tissue, between the vagina and urethra, which had burst into the urethra, and had never healed properly from the bottom.

#### CASE XXXIV.

*Complete prolapsus uteri in an unmarried woman brought on by strain. Operation. Cure.*

UNDER MR. GREIG SMITH.

J. R., æt. 29, cook. 49 days.

While lifting a heavy pot off the fire patient felt something give way in the lower part of the abdomen, and at night, when in bed, she discovered a smooth, hard, rounded tumour protruding half-an-inch through the vulva. This had happened two years before admission, and in the meantime her condition had become much worse, more than two inches of the swelling sometimes being protruded. The uterine cavity was three and a half inches long, and the uterine tissue was correspondingly hypertrophied; the os uteri was somewhat abraded; menstruation was very profuse and painful. The prolapse seemed to depend on yielding of the posterior and anterior walls of the vagina equally. The patient being under the influence of dichloride of ethidene, Emmet's operation for procidentia was performed. Eight days after operation the silver sutures were removed, and the incisions were found to have firmly united. The patient



was confined to the supine position a fortnight longer, and astringent injections were daily administered. A small ring pessary was worn for a few weeks before she was dismissed.

#### CASE XXXV.

*Retroversion of gravid uterus. Retention of urine and fæces for a week. Replacement by hand in rectum. Abortion. Recovery.*

UNDER MR. GREIG SMITH.

R. N., æt. 24, married woman. 15 days.

A week before admission, while straining at stool, patient was suddenly seized with a severe pain in the back and lower part of the belly. The pain had continued on the increase up till admission, and during this time she had been unable to pass either urine or fæces. When seen she was in a very dangerous condition, collapsed and almost pulseless. A tumour, evidently with fluid contents, filled the whole of the abdominal cavity, and pressed downwards the anterior vaginal wall so much as to cause a very perceptible bulging of the perineum. Per rectum, the uterus was with some difficulty made out, enlarged to the size of a five or six months' pregnancy, pushed to the left side, retroverted and firmly blocked in the pelvis. All attempts at reduction in the genu-pectoral position by the fingers having failed, the whole hand was introduced into the rectum and the uterus replaced. More than a gallon of putrid blood-stained urine was withdrawn. The bladder was washed out with a weak warmed solution of carbolic acid, and a simple laxative enema was administered. Twelve hours afterwards, without any trouble, she aborted a fœtus of

about five months. There was no incontinence of fœces, but incontinence of urine continued for six weeks. Thereafter she was quite well.

## CASE XXXVI.

*Vaginismus. Prolapse of rectum. Conical os uteri. Operation. Cure.*

UNDER MR. GREIG SMITH.

E. E., æt. 28, married nine years. 120 days.

Patient always had severe pain at her menstrual periods, but was fairly regular till marriage, when they came on with greater frequency and lasted longer. When she was married, at nineteen, she fainted away on the first attempt at copulation, so painful was the process. Coition had never been performed, the slightest attempt bringing on spasms so violent and painful that she screamed out. Within the last four years the condition of spasm became so much aggravated that the slightest irritation, such as a sharp movement of the body, a gust of cold air, or a mental surprise would bring one on. Latterly she was having them very frequently. It was impossible to introduce the forefinger. An attempt to do so brought on violent contraction of the sphincter, and made the patient scream out. She had had continual medical advice at London and provincial hospitals and from private practitioners, but had derived no benefit therefrom. On admission she denied ever having had any trouble from her bowels or bladder; indeed, she thought she was very healthy with the exception of her menstrual and vaginal troubles. While she was under chloroform a careful examination of the bladder, rectum, vagina, and uterus was made. On drawing the finger

out of the rectum the mucous membrane, which was very lax and thin, prolapsed to the extent of an inch. Except that the os was conoid, the uterus was healthy. Nothing further abnormal was detected. She was not aware of having prolapse of the rectum, but on examination this was found to take place after every motion, returning of itself as she stood up. The ordinary treatment by astringent and acid applications having failed, three semilunar pieces were longitudinally removed from the rectal mucous membrane. The longitudinal fold was pinched up, double stitches inserted, and the protruding portion cut off with scissors. In this way hæmorrhage was avoided. A complete cure of the prolapsus recti resulted. Rest in bed, the occasional passing of a uterine sound, several dilations of the vagina, and the administration of the bromide of iron was the further treatment pursued. She went out completely cured of her condition of vaginal spasm; and, when last seen (three months after dismissal), said that coition was effected without difficulty.

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## SHORT NOTES OF INTERESTING MEDICAL CASES.

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### CASE I.

*Aneurism of basilar artery. Rupture. Death.*

UNDER DR. SHAW.

Charles O., æt. 32.

Patient, who was a seaman, was brought to the Infirmary quite dead. His companions stated that he had been drinking heavily some time, and that he had been seized with violent sickness and diarrhœa two days before death. About an hour before his admission he was trying to do some work on the deck of his ship, when he suddenly fell down, and scarcely moved again.

*P. M. E.*—Body of a very well-nourished and muscular man. Aneurism of basilar artery, rather larger than a pea, which had ruptured, and the blood had become extravasated over the medulla and into the ventricles. Not much disease in the other vessels.

### CASE II.

*Hemiplegia coming on after a blow on head. Delirium and coma. Death. Large clot in brain.*

UNDER DR. SHAW.

George W., æt. 56. 3 days.

Three days before admission the patient received a

blow with the fist on his right temple. About ten minutes afterwards he fell off his chair, quite unconscious, and remained in that condition for several hours. When he recovered consciousness, it was found that he was quite paralysed on the left side, and his speech was very indistinct. On admission, he was conscious, but stupid and rather incoherent. Speech slow and thick. Left arm completely paralysed. Could move leg a little. Face drawn to right side. He complained of severe pain in his head. No cardiac bruit. No albuminuria. Slight bruise on right temple. During the two days after admission he was very restless and delirious. He then gradually sank into coma, and died on the third day, without any convulsions.

At the *P. M. E.* no fracture of the cranium was found. There was some ecchymosis in right temple and round right orbit. No inflammation of meninges. No effusion into ventricles. Large hæmorrhage in white matter of right hemisphere, tearing up the extra-ventricular portion of the corpus striatum and the external portion of the optic thalamus. The heart was rather hypertrophied. Kidneys healthy.

#### CASE III.

*Meningitis, cortex lesions, hemiplegia, following sunstroke.*

UNDER DR. SHINGLETON SMITH.

James V., æt. 27, married.

Illness commenced with an attack of vertigo, supposed to be "sunstroke;" two days after vomiting commenced, and he felt throbbing pain in the head; twelve days later tingling in the left arm and leg was noticed, and then numbness with gradual loss of power commencing in the left arm and extending quickly to the leg.

On admission, July 17th, complete motor paralysis of left arm, with partial paralysis of left leg. Sensation slightly impaired. Pulse 88; temp. 101.8°.

Two days after a violent convulsive attack, bilateral, and accompanied at the onset by a low cry, was observed; a second attack followed within two hours. After the second attack the temperature was found to be 106° Fahr. It was observed that the convulsive movements did not affect both sides equally, the head was drawn to the left, the left angle of the mouth twitched, and the left limbs were much more affected than the right. The patient gradually became comatose, with flaccidity of the left limbs: the temperature continued high, the pulse became weaker, and he died on July 22nd.

*P. M. E.*—A patch of yellow lymph, quarter of an inch in thickness, covered the following convolutions, ascending frontal, ascending parietal, parts of first and second frontal, and superior parietal, on the right side: the pia mater on left side was slightly opaque from the presence of a thin layer of lymph. No tubercles were present in brain or in lungs. A large ulcer existed in the left auricle, and embolic patches existed in the spleen and left kidney. Numerous petechial spots were seen on the skin, pericardium, and endocardium.

#### CASE IV.

*Hæmorrhage into corpus striatum. Independent hæmorrhage into pons varolii. Death.*

UNDER DR. WALDO.

Mary Ann M., æt. 44. 1 day.

About 10 a.m. on day of admission patient was found lying on the floor of her room in an unconscious condi-



tion. She had spoken to some friends about half an hour before, and seemed then in good health. When seen, she was quite unconscious; breathing slow and stertorous, with right cheek puffing out during expiration. Left pupil dilated, right rather small. Face drawn slightly to left side. Right arm and leg fell more heavily than left when raised. When soles of feet were tickled both arms and legs moved a little. Muscles of left leg seemed firmer than right. She muttered a little at times. No cardiac bruit. No œdema. Urine drawn off with catheter, and found to contain a large quantity albumen, but no tube-casts found. About 1 p.m. it was noticed that her face had become symmetrical; there was drooping of the angles of the mouth, and both cheeks seemed quite flaccid. No puffing out in expiration. Breathing not now stertorous, but quiet and slow. Limbs all fell quite limp when lifted, and seemed equally paralysed. Still moved limbs a little when feet were tickled. Pupils same as before. No further change in symptoms. She gradually sank and died about 4 p.m. She was a patient in the Infirmary about eight months before, with paralysis of left arm and leg. Her left pupil was then larger than right. She had no albuminuria at that time. She recovered power of limbs almost completely.

*P. M. E.*—Large, recent clot in left lateral ventricle, tearing up corp. striatum and optic thalamus, and extending into white matter for considerable distance. A little blood in right ventricle (passed through). Recent hæmorrhage, rather larger than a cherry-stone in middle of pons, stretching across median line. Small, old hæmorrhage posterior to this, on left side of pons. Old cicatrix in right corp. striatum. “Horse-shoe” kidney. Hypertrophy of heart.

## CASE V.

*Hæmorrhage into optic thalamus. Paralysis of arm and leg, but not of face. Acute œdema of lungs. Death.*

UNDER DR. WALDO.

Patrick H., æt. 40. 1 day.

Patient had always been healthy. Had drunk pretty freely. On evening before admission he complained of "a cold," and was rather restless during the night. Went out to go to his work, but fell down in the street and was brought to Infirmary.

*On admission*:—Perfectly comatose. He winces a little when forehead is struck sharply. When legs are pricked with a pin he moves them both a little, the left more than the right, and the latter also falls more limp when lifted. He moves left arm a little when irritated, but no voluntary movement of right arm; occasional twitchings in right arm; face *not* twisted; no cardiac bruit; pulse slow and full; no œdema of extremities; pupils small and fixed; breathing quiet, not stertorous.

He continued in that condition for eight or nine hours, the paralysis gradually becoming more complete and the pupils smaller. The breathing then became more rapid and difficult, and frothy serum came from mouth. The pulmonary œdema rapidly increased, and he died in about eleven hours from admission.

*P. M. E.*—Clot occupying position of lower part of left optic thalamus; upper part of optic thalamus and corpus striatum not injured; blood in left ventricle and blood-tinged serum in right; blood had passed through "iter a tertio" and lay on floor of fourth ventricle; vegetations on mitral valve; kidneys congested, otherwise healthy.

## CASE VI.

*Tubercular meningitis. Death in first convulsion.*

UNDER DR. SHINGLETON SMITH.

Louisa F., æt. 15.

Patient's friends stated that she had been complaining of sickness and giddiness for about four days. She had taken a few pills and a seidlitz powder, prescribed by a chemist, and had continued to go about her work until the day of admission, when she felt so giddy that she lay in bed. She was put into a cab to come to the Infirmary, but on the way was seized with convulsions and became insensible. When seen she was perfectly comatose, and could not be roused. Œdema of the lungs set in, and she died within half an hour after admission.

*P. M. E.* revealed the pathological appearances of tubercular meningitis in a very early stage; not much lymph, but considerable thickening of the coats of the smaller arteries, with proliferation of nuclei, was seen with the microscope.

## CASE VII.

*Tubercular meningitis. Tubercle in liver and spleen.*

UNDER DR. SHINGLETON SMITH.

H. P., æt. 45, labourer.

Previously in good health, was seized with severe pains in stomach and vomiting after all food at the end of March, 1879. Vomiting lasted only four days. Pain persisted, more or less, always worse immediately after food and lasting several hours; much flatulence; bowels always fairly regular. A good deal of headache at times; a good deal of cough. Kept his bed for four weeks.



Then got better, and was able to be up and about a little daily for about ten weeks, when he had to take to bed again. For three weeks before admission severe pain in legs and thighs, worse at night. Admitted to Infirmary, July 16th, 1879. Then, well-nourished and muscular; expression anxious; severe pain and tenderness at epigastrium, passing through to back; severe vomiting (especially after food) of liquid contents of stomach without bile; tongue, clean and moist; severe pain in head, chiefly frontal; sleep disturbed, waking with start and delusions; occasional giddiness and flashing before eyes; severe pain in legs and thighs, with muscular tenderness; little cough and no expectoration; optic discs and retinæ normal; lungs, heart and abdominal organs gave no indications of disease. Vomiting, pains in head and legs and disturbed sleep continued, temporarily relieved by chloral and bromide of potassium. Became delirious on the third day. Drowsy and heavy on the fourth day; unable to pass water; urine drawn off by catheter was normal. Vomiting abated somewhat; delirium alternated with coma; urine passed involuntarily; coma gradually deepened; death on eleventh day. Temperature throughout not over  $101^{\circ}$  (for five days under  $100^{\circ}$ ); pulse averaged about 100.

*P. M. E.*—Meninges of convexity injected; increase of cerebro-spinal fluid; deposits of lymph (fibrinous in parts) over posterior base, extending into Sylvian fissures and to superior aspect of cerebellum; vessels at base atheromatous; pia mater at base studded with millet-seed granules, chiefly along vessels of Sylvian fissure and posterior chorioid arteries, much thickened and in parts adherent to brain; brain rather pulpy; anterior cerebral lobes adherent in fissure; lateral ventricles distended with excess of

fluid; choroid plexuses beady and cystic; under microscope, the walls of smaller vessels were thickened and shewed much nuclear proliferation. Bases of both lungs hypostatically hyperæmic; no tubercle; bronchial glands normal. On mitral and aortic valves and on aorta, atheromatous patches; heart otherwise normal. Under Glisson's capsule, scattered irregularly, numerous small grey and yellow nodules; similar nodules here and there in substance of liver; liver hyperæmic; under microscope, nodules appeared to be tubercle. Spleen had numerous millet-seed granules in substance; under microscope, similar to those in liver. No tubercle or disease in any other organ. No caseation anywhere.

#### CASE VIII.

*Chronic hydrocephalus, with tumours in cerebellum.*

UNDER DR. SHINGLETON SMITH.

Frederick H., æt. 8.

Symptoms of eight months' duration, pain in head, giddiness and difficulty in walking. Loss of sight two months later. Three months after convulsive attacks occurred, sometimes five or six "fits" in a day. Right hemiplegia followed; occasional vomiting and some defect in hearing were observed.

Oct. 12th, 1878.—Emaciated; semi-conscious; right hemiplegia; unable to see, talk, or do anything for himself; smell and hearing defective; both pupils dilated and insensible to light; retinal veins distended, arteries small, the disc indistinguishable.

Nov. 8th.—The head was observed to be much retracted. There had been no further convulsions or vomiting.

Feb. 6th.—He had continued to take food, but emaciation had been steadily progressive. Flexion and rigidity of limbs continued, and there was extreme retraction of the head. Bed-sores had formed on all prominent points in spite of all precautions. Death took place twelve months after date of attack.

The lateral ventricles were found to be distended to five times their normal size. The cerebellum contained two large tubercular masses: the left lobe was enlarged, very hard to the touch, and contained a tumour as large as an egg, yellow, firm, and concentrically marked on section; the right lobe contained a similar mass, but smaller.

The lungs were infiltrated with miliary tubercles, and the bronchial and mesenteric glands contained much caseous matter.

#### CASE IX.

*Acute myelitis, probably caused by cold. Good effects of ergot.  
Death.*

#### UNDER DR. SPENCER.

W. B., æt. 35, coachman and gardener.

Always healthy and temperate; never had syphilis. For some time before present illness had been accustomed daily to work in garden (on a cold, damp, clayey soil) whilst still wet and chilled about feet and legs from washing carriage. Had had lumbar pain for a month when, on Dec. 9, 1877, “stinging” across toes of right foot, “deadness” in that foot were noticed; these sensations extended up the leg, and he lost power in it, and in ten days from their onset, he had to give up work. Then began difficult micturition. A week later, retention of



urine (catheter); right leg affected to hip and groin; the early symptoms had appeared in left foot. Admitted to Infirmary on Dec. 29, 1877. Then, stinging and numbness from toes to hip, and complete loss of power in right leg; sensation much impaired; reflex action not abolished. Similar state of things, to lesser degree, in left foot and lower leg. Pain in both groins, worse at night. Severe pain across loins; very sensitive to hot sponge over lower dorsal and lumbar vertebræ, and laterally for some distance. Complete retention of urine. Obstinate constipation. No other affection of organs or functions could be detected. Florid and robust in appearance. Treatment by blisters and iodide of potassium. On Jan. 7, 1878, had had no action of bowels for five days in spite of measures for relief; was unconscious of enemas. Belladonna (ext. gr.  $\frac{1}{4}$ ) was given every two hours for 48 hours; bowels acted involuntarily several times in the course of the next 24 hours. Now, complete paralysis of both legs; legs feel as if constantly moving up and down; sensation lost absolutely below a line round body at level of umbilicus; abdomen soft, not distended, resonant; no pain or tenderness anywhere. Treatment with ergot (ext. liq. ʒss. every six hours) commenced on Jan. 9; iodine paint over lumbar spine. Jan. 17, much better in himself; pain in loins severe at times; constipation obstinate; urine began to dribble away to-day. Next day, the limit of loss of sensation was found to be three inches lower than that of Jan. 9th. Tongue clean; takes food well. By Feb. 1st, sensation regained over whole abdomen and somewhat in femoral region; continued at this lower level to time of death. Bed sores formed early in the case and healed during the use of ergot, recurring later on. Ergot discontinued on Feb. 18th. Strength

gradually failed, diarrhœa supervened, and death on March 27th.

*P. M. E.*—Spinal cord, opposite last dorsal and first lumbar vertebræ, diffluent and puriform; soft, creamy, and decreasingly puriform in centre, for some distance above and below; membranes opposite lesion were much thickened and adherent; the lesion was traced to upper dorsal and low lumbar regions under microscope. All other organs (except bladder, which shewed effects of the cystitis) were healthy.

#### CASE X.

*Disseminated cerebro-spinal sclerosis. Treatment by belladonna, toxic symptoms, but no diminution of tremor.*

UNDER DR. SHINGLETON SMITH.

Stephen B., æt. 34, haulier, married.

Had good health till two years ago. An attack of giddiness, thought to be "sunstroke," was followed by tremor of right leg and arm. Two similar attacks occurred subsequently, the tremors increased, and the patient gave up work three months ago.

On admission, April 22, 1878, his mental condition was defective, and articulation indistinct; he answered coherently, but was disposed to laugh constantly, and spoke slowly and with effort. Constant tremor of facial muscles noticed when speaking. No general tremor when at rest, but violent spasmodic contractions were brought on by any voluntary movement; was able to stand and walk without assistance, but gait was ataxic with tremor super-added. Had great difficulty in getting into the erect position, and violent bilateral jactitation followed any attempt to feed himself. There was no loss of mus-

cular power, and no wasting of muscles. Sensation unimpaired. Sight was good, but the optic discs were greyish, and the vessels small. His general health and nutrition were good.

Belladonna was given in doses of fifteen minims of the tincture three times daily: twelve days later patient was delirious, refused his food, and would not get up; pupils were dilated widely.

The day following the discontinuance of the belladonna the delirium had subsided, he took food again, and was able to walk, but the tremor had been in no respect affected by the action of the drug.

#### CASE XI.

*Spasmodic rhythmical contractions of diaphragm, recti, and other muscles. Ultimate recovery.*

UNDER DR. SHINGLETON SMITH.

Laura F., æt. 20, single, servant.

Admitted Sept. 10, 1878, for peculiar choreic jactitation, which came on the previous evening, kept her awake all the night, and persisted in the morning. A similar attack six months previously subsided after two nights and one day. Patient was well nourished, but anæmic, with a tendency to globus, sedate looking and unexcitable, catamenia regular. The body was being constantly jerked by spasmodic contractions of the recti abdominis and diaphragm, the contractions being regular at about 70 per minute. They ceased entirely during sleep, but recommenced immediately on waking.

Sept. 28, the contractions were more vigorous than before, and had extended to the sterno-mastoid muscles: the rate had increased to 140 per minute. After twenty-



eight days the spasms ceased suddenly without manifest cause, but four days later re-commenced in the recti, and afterwards became more general: the recti, sterno-mastoids, scaleni, pectoral muscles, and the diaphragm were all affected, as also the biceps and triceps brachii. After two months slow but steady improvement commenced, and on Jan. 22, 1879, the patient was sent home quite well. Nine months afterwards she applied as an out-patient with dyspeptic symptoms, and there had been no return of the muscular spasms.

## CASE XII.

*Hemi-anæsthesia. Pneumonia. Hyperpyrexia. Death.*

UNDER DR. WALDO.

Henrietta J., æt. 21. 283 days.

Patient had been out of health for fully a month before admission. She had suffered a good deal from headache and sleeplessness, and for about ten days she had had a sore throat and obstinate diarrhœa. Vomiting had been rather troublesome; menstruation too profuse. She had led a loose life for some time, but had been living more quietly latterly.

*On admission*:—Well nourished; nervous and excitable; breathing jerky; abdomen tender; tonsils enlarged; temperature, about 103°. In a few days her throat became very much better, and her temperature fell to normal. She began to complain then of pains in her left leg. She had a chronic “housemaid’s knee” on that leg. Blisters were applied to it. The pains, however, increased in severity, and the leg became so weak that she could scarcely put any weight on it. She suffered a good deal from pains in right frontal region at times. Men-

struation continued profuse. She went on in this way for about three months, the leg getting gradually weaker, and the left arm also losing strength. She had no fits of any kind, but was very nervous and depressed. She then began to complain of numbness on the left side and in the left arm and leg, and on touching her on any part of the left side sensibility was found to be impaired. This got gradually worse, until there was complete hemianæsthesia, affecting left side of scalp, face, tongue, neck, trunk and left limbs. Pins could be stuck into almost any distance without making her wince, and the punctures scarcely ever caused bleeding. Even the nipple was quite insensitive, and also the mucous surfaces, on the inside of the mouth, and the vagina on the left side. She had great pain and tenderness in the region of the right ovary. Frontal headache continued very severe. Menorrhagia persisted. She did not sleep well, and took very little food, but her nutrition kept up pretty well. She became peevish and irritable, and had delusions of suspicion regarding her fellow-patients. Numerous drugs were tried, but all failed to give her anything like permanent relief. Experiments were made as to the effects of discs of metal, &c. (Metalloscopy). Discs of wood and the following metals—gold, silver, copper, iron, zinc and lead—were bandaged to the leg and left on for some time. Slight temporary return of sensation seemed to follow the application of copper and iron, but the results were inconstant and unsatisfactory. She became gradually weaker, physically and mentally. About a week before her death an attack of hypostatic pneumonia came on, and her temperature rose. She could scarcely swallow any food; cough was very troublesome. She became semi-comatose, and continued so for two or three days.

Just before her death her temperature rose to nearly 107° F.

At the *P. M. E.* the lungs were found very much congested, with consolidated patches here and there. No lesion could be found in any part of the nervous system.

### CASE XIII.

*Morbus Addisonii. Progressive asthenia. Syncope. Death.*

UNDER DR. WALDO.

Henry L., æt. 48. 18 days.

Patient had been out of health for about six months before admission. His appetite had been poor, and he frequently had attacks of vomiting soon after taking food. He had never vomited any blood. Had been gradually losing flesh and getting weaker. He had also noticed that his skin was getting darker in colour. Slight cough. He had had ague sixteen years before the time when he came under observation, but no recurrence of it since; rheumatic fever four years after that; and after another five years his left elbow joint had been excised for disease. It continued to discharge for a long time, but with the aid of a leather apparatus he could use it very well. When examined, he was found to be very thin and anæmic. He had a general bronzing of the skin of the whole body, but no deeply pigmented patches, except bluish patches on inside of cheeks. Circulation was very feebly carried on; pulse small; a few rhonchi in lungs; unable for any exertion; slight pain in epigastrium; no albuminuria. While under treatment he gradually became weaker. No change in colour of skin. He had scarcely any vomiting for fully a fortnight after admission, then it



came on severely again. No other acute symptoms developed themselves. He died suddenly in syncope.

*P. M. E.*—Both supra-renal bodies were enlarged, and had lost their normal shape. The left one was completely transformed into a caseous mass. The right one was of a greyish-yellow colour, tough and fibrous, and presented several caseous spots here and there. Microscopically, extensive transformation of tubules into fibrous tissue. Kidneys healthy. Caseous bronchial and mesenteric glands, and a small cavity, containing caseous matter, in the spleen. Heart small and fatty. Old phthisical signs at apex of right lung. Liver rather fatty.

#### CASE XIV.

*Morbus Addisonii (?)*. *Marked improvement.*

UNDER DR. WALDO.

Charles B., æt. 27. 64 days.

Had a fever when fifteen years old, and had not been so strong since. His skin had gradually got darker in colour since that time. Had not had any cough, nor had he lost flesh. He had suffered a good deal from shortness of breath. Had been getting gradually much weaker and less fit for work during the year preceding admission. When he came under observation he was found to be ill-nourished. His skin all over had a bronzed tint, and in the flexures of his joints and in the backs of his hands there were patches of dark brown pigmentation. Several bluish patches in the mouth, on palate, cheeks and gums. He was very anæmic. Pulse small and compressible. Systolic bruit heard in all the cardiac areas, harsher at base. Dyspnœa and palpitation on the slightest exertion. The urine did not contain albumen, sp. gr. 1012.

He was kept quiet in bed for several weeks, and treated with cod liver oil, lacto-peptine and tonics. He gradually gained strength, and became stouter and much less anæmic. Pulse improved greatly. Dyspnœa on exertion diminished very much. Appetite and digestion became much better. Shortly after going out he was able to return to his occupation of brickmaking. He was under observation as an out-patient for a long time, and continued to keep pretty well.

## CASE XV.

*Morbus Addisonii* (?). *Asthenia*. *Improvement*.

UNDER DR. SHINGLETON SMITH.

Alice K., æt. 15. 40 days.

Patient had been in delicate health for a good many years, and had been gradually growing weaker. She had suffered a good deal from pains in legs, which had been swollen a little at times. She had also had persistent pain in left side. Had only menstruated once. Had not had any cough. Appetite had been fairly good. Had suffered from giddiness and occasional attacks of syncope. About a year before admission she noticed the skin of her neck becoming of a brown colour. This had grown darker, and the skin of arms, legs, and body had also become affected. She had suffered a good deal from itching of the skin.

On examination, she was found to be fairly well nourished, and well developed for her age. She was anæmic. Skin of neck was of a brown colour; dark patches under eyes; large areas of pigmentation in axillæ, extending down sides of thorax, in the form of dark circles arranged vertically edge to edge. Large pig-

mented patches with intervening normal skin over front of thorax and abdomen; none on back; the pigmentation was well marked in groins and thighs, fainter on legs, and again more distinct on insteps. Arms slightly pigmented; bluish tinge on roof of mouth. Lungs normal. No cardiac bruit; pulse rather compressible. No albuminuria. While under treatment she suffered a good deal from palpitation, giddiness, and itching of skin. Iron, arsenic, &c., were administered, and she went out greatly improved.

#### CASE XVI.

*Chronic valvular disease of heart. Intercurrent attack of acute endocarditis. Death.*

UNDER DR. WALDO.

William C., æt. 18. 66 days.

Patient had attack of acute rheumatism about three years before admission, and had not been strong since. Had suffered from dyspnœa and palpitation, and occasional pains in limbs. Hæmoptysis once or twice. No œdema of extremities. Had been off work for three weeks. On admission, he was thin and anæmic. A loud, harsh, systolic bruit was audible at apex of heart, and round to back; also systolic and diastolic aortic bruits. Pulse very jerky and collapsing. Lungs resonant, a few sibili, cough. No œdema or ascites. While under observation he did not make satisfactory progress at all. For a time, he seemed to improve a little, then he fell back again. Palpitation was very severe at times, and he frequently had articular pains, with a little swelling. He was treated with digitalis, arsenic, iron, &c. After he had been in the wards for nine weeks he became sud-



denly worse, had very severe pains in chest, great dyspnœa and palpitation, and the temperature rose very high. There were abundant dry and moist sounds in the lungs. The pulse was very rapid. The mitral systolic bruit was louder and harsher than before. He became of a pale, ashy colour, was very restless, and at times quite unconscious. He grew rapidly worse, and died on the third day.

*P. M. E.*—On the posterior cusp of the mitral valve was a rough, circular patch about the size of a shilling, and on this was deposited recent fibrinous vegetations. This was partly on the auricular wall, and partly on the valve itself. Base of valve rough and thickened. The muscoli papillares were hypertrophied, and three or four of the chordæ tendineæ ruptured, the ends being ragged. Left ventricle hypertrophied. Vegetations on aortic valves, the cusps of which were thickened. Spleen was enlarged and congested, and contained a pretty large infarct. Lungs engorged, portions collapsed.

#### CASE XVII.

*Cronic valvular disease of heart. Intercurrent acute endocarditis. Syncope. Death.*

UNDER DR. SHAW.

Melinda S., æt. 25. 62 days.

Had acute rheumatism about eight years before admission; made good recovery; no recurrence. Breath short on exertion for about a year; severe palpitation two months; short, dry cough. Slight œdema of feet and legs occasionally. Pain in left side.

*On admission*—extremely anæmic. Apex beat thrown

down and out ; diffuse cardiac impulse ; regular, but rapid action ; slight thrill on palpation ; loud, harsh blow at apex, partially presystolic and partially systolic in rhythm ; systolic bruit at base. Pulse rapid and compressible, rather jerking. Lungs resonant, scarcely any cough. No albuminuria.

While under observation her progress was very unsatisfactory. Her temperature rose every evening, frequently very high. She had occasional articular pains, but very little swelling. Præcordial pain and palpitation were frequently very severe. Profuse sweatings often at nights, appetite very poor. Albumen appeared in the urine. Pulse became much softer and more compressible. Her strength gradually failed, and she died in syncope.

*P. M. E.*—On the posterior wall of the left auricle was a circular patch, about the size of a florin, rough and covered with recent fibrinous vegetations, and recent clot adhering to it. Mitral valve thick and rough. Left ventricle hypertrophied. Vegetations on aortic valves ; pale clots in cavities of heart and in great vessels. Lungs rather congested. Spleen very large, congested, rather soft, and containing five or six embolic patches of different colour and consistence, some of them very large. Liver fatty. Kidneys large, and a small infarct in one of them.

#### CASE XVIII.

*Congenital pulmonary stenosis. Embolism of right brachial artery. Improvement.*

UNDER DR. SPENCER.

Stephen M., æt. 12. 49 days.

Had always been delicate. Blue colour of face and

lips since he was 15 months old. Extremities of fingers and toes had always been clubbed. Great palpitation and dyspnœa on exertion. Not much cough. Swelling of feet and legs at times. About a week before admission he had suddenly lost the use of his right arm to a great extent. It became numb, swollen, and painful. Some of the power had since returned. On admission he was found to be badly nourished. Marked cyanosis of face and lips. Fingers and toes very much clubbed at ends. Very distinct præcordial thrill. Apex-beat thrown outwards. Loud, harsh systolic blow in pulmonary area, and transmitted to the other areas. Pulmonary second sound very greatly accentuated. Palpitation and dyspnœa on the slightest exertion. Some dulness, harsh breathing, and increased vocal resonance at apex of right lung. Right arm rather swollen, numb, and painful, and radial pulse rather smaller than left. Grasp of hand defective. Under rest and tonic treatment he improved considerably. Palpitation and dyspnœa became much less troublesome. The right arm returned to its usual condition. The cyanosis of the face diminished a little. The pulse improved in character.

## CASE XIX.

*Chronic rheumatism and emphysema. Weak heart.*

*Gangrene of both legs. Death.*

UNDER DR. WALDO.

Harriet V., æt. 41. 32 days.

Patient had had four attacks of acute rheumatism, the first at fifteen years of age. She had also had frequent pains in her joints, chiefly ankles. Cough and shortness of breath for several winters. Appetite and



digestion had been good, and she had kept very stout. Had had pains in joints for several weeks, but they had come on much more severely four or five days before admission. She was "flat-footed"; ankle-joints considerably thickened; knees painful; chest emphysematous; heart's action very weak; pulse small and compressible; extremities cold. Cough and dyspnœa gradually became worse. Left base became consolidated, and there were some rather fine crepitations. This did not clear up. Numerous râles were heard all over the chest, but she soon lost the power of expectorating. Pains in knees, ankles and feet increased in severity. About a fortnight after admission dry gangrene set in in both feet, and gradually spread up to nearly the middle of the thighs. No distinct line of demarcation. Circulation became gradually weaker, in spite of the free use of stimulants, and she died of exhaustion.

*P. M. E.*—Firm clot in abdominal aorta astride the bifurcation, and extending down into iliacs and femorals on both sides. Inner coats of arteries very much congested and roughened. Heart dilated, soft and fatty. Lungs emphysematous and œdematous, but not containing embolic patches. One small embolus in spleen and several in kidneys.

#### CASE XX.

*Acute Bright's disease. Uræmia. Epileptiform convulsions.  
Œdema of lungs. Death.*

#### UNDER DR. SHAW.

Henry O., æt. 31. 10 days.

Patient had been very much exposed to the weather for several weeks while tramping about the country. He

had felt rather out of health for a fortnight; his legs had felt stiff and painful, he had shiverings, and he noticed that he was passing less water than usual, and it was of a high colour. Slight cough for a few days. No pain in back. Breath rather short. Legs swollen a little.

*On admission*—his legs were œdematous, tense and glazed; no ascites. Face rather puffy. Urine very smoky; large quantity of albumen; much epithelium, and a few epithelial hyaline casts. Heart's action quiet. No bruit. Evening temperature slightly raised. Wheezing in chest. Cough troublesome. For the first seven days after admission he seemed to be going on pretty well; the quantity of urine kept up to about 40 oz., or nearly so, with a specific gravity of about 1015; the blood diminished a little, but the quantity of albumen continued very large. Swelling of legs kept about the same. He had slight pain in his back. On the eighth day he passed about 46 oz. of urine, but the specific gravity had fallen to 1008, and it contained more albumen. He complained very much of pains in his head, and in the evening he had an epileptiform convulsion. During the night he had several convulsions, and on the following day he became comatose, and continued in this condition, with occasional convulsions, till the morning of the next day, when he died. Pulmonary œdema set in a few hours before death.

*P. M. E.*—Kidneys very much congested. Cortex large. Vessels of pia mater engorged. Brain hyperæmic and soft. No fluid in ventricles. Lungs congested and œdematous.



## CASE XXI.

*Chronic Bright's disease. Purpura. Epistaxis. Suppression of urine. Uræmia. Death.*

UNDER DR. WALDO.

Thomas Hill, æt. 30. 10 days.

Patient had been in delicate health for a considerable time, much worse during the three months preceding his admission. He had had frequent attacks of bleeding from nose and mouth; blue spots on legs for about three months; daily quantity of urine varied greatly, but had been getting less lately. He was very anæmic and pasty-looking. Eyelids puffy. Legs rather œdematous. Copious purpuric eruption on both legs and a few spots on arms. No cardiac bruit. Pulse rather tense. He was drowsy and stupid. Had purulent discharge from left ear. Urine small in quantity and contained a large amount of albumen. After admission he had repeated attacks of epistaxis and frequent sickness and vomiting. He passed very restless nights. Had troublesome diarrhœa. The quantity of urine diminished and the proportion of albumen increased. He was treated with diuretics, digitalis (by mouth and as fomentations) and hot-air baths. Suppression of urine gradually set in, however, and he became uræmic. Eight days after admission he had almost complete suppression, and he was quite delirious. Breathing loud and puffing. The purpuric spots had become very much fainter. The suppression and delirium continued, he became gradually weaker, and died two days after. No *P. M. E.* could be obtained.



## CASE XXII.

*Contracted granular kidneys. Epileptiform convulsions.  
Death.*

UNDER DR. SHAW.

Eliza J., æt. 35. 31 days.

Had suffered from occipital and vertical headache, more or less severe, for about two years. Thought it was caused by a mental shock she received. Had an epileptiform convulsion about four months before admission, and since that time they had recurred very frequently, sometimes as many as eight or nine in one day. Memory has been impaired and sight rather dim. Passing large quantity of urine. On admission she was found to have considerable cardiac hypertrophy. Passed large quantity of urine, which contained a great deal of albumen and granular tube-casts. No œdema. She had frequent attacks of severe pain in the head while under observation. Quantity of urine kept very large. She had no return of the fits till the day before her death, when she had slight epistaxis and an epileptiform convulsion soon after. This was followed by many more during that day and the next night, and she gradually became comatose and died next morning.

*P. M. E.*—Small, hard, granular kidneys. Capsule adherent. Several urinary cysts on surface. Cortex diminished. Walls of left ventricle very thick. Central veins full. Remains of old hæmorrhage at posterior part of right temporo-sphenoidal lobe.

## CASE XXIII.

*Acute desquamative nephritis. Uræmic convulsions and delirium. Recovery.*

UNDER DR. WALDO.

Alfred R., æt. 14. 68 days.

About a fortnight before admission patient felt ill and had a violent shivering. Shortly afterwards he noticed that he was passing less water than usual, and his feet and legs became swollen and his face puffy. Very much exposed to cold at his work in a blacksmith's shop. On afternoon of admission his friends heard peculiar sounds coming from room where he was, and on going in found him convulsed and struggling violently. After the fit passed off he continued very restless. When seen he was quite unconscious, struggling, tearing his clothes, &c. Face and legs œdematous. Pupils rather large. Skin hot and dry. Pulse rapid. Shortly after admission he had several epileptiform convulsions, and became extremely violent and abusive. Chloroform was given to quiet him; croton oil administered, and the hot-air bath employed several times during the evening. When he became a little quieter chloral was substituted for the chloroform. After the bowels had acted freely he became rather more conscious. Temperature rose above 103°, and remained there for several hours, then gradually fell. Urine drawn off with the catheter was found to contain a good deal of blood and numerous tube-casts. A very small quantity secreted. Infusion of digitalis was given in ʒss. doses every two hours at first, and afterwards less frequently. The next day he passed 28 oz. of urine. Had

passed quiet night, and was quite rational. The two following days he passed 72 and 80 oz. of urine, and for the next month it varied from 55 to 65 oz. The quantity of urine was very large at first, but gradually diminished, and the dropsy disappeared. His subsequent progress towards recovery was uninterrupted. When he left the Infirmary there was still some albumen in his urine, but it gradually diminished, and he has enjoyed good health since.

## CASE XXIV.

*Cholæmia. Convulsions. Coma. Death. Ascaris in intestine.*

## UNDER DR. SPENCER.

Charles S., æt. 5 years. 1 day.

The day before admission the child became sick, had several convulsions, and at night he became delirious. When seen he was deeply jaundiced, the abdomen was distended and tympanitic, and his pulse very rapid and feeble. He was delirious when admitted, and had several convulsions soon after. He vomited up some dark-coloured blood in the evening. In the night he became comatose, and continued in this condition till 6 p.m. next day, when he died.

At the *P. M. E.* an ascaris, four inches long, was found lying in the small intestine, and from two to three feet below this the mucous membrane was covered with slightly altered blood for a distance of about two feet. No abrasion discoverable. Liver weighed 18½ oz. Substance normal. Bile-ducts healthy. Gall-bladder full of fluid bile. A little fluid in peritoneum. Serous membranes bile-stained. Brain and other viscera healthy.



## CASE XXV.

*Cirrhosis of liver. Hæmorrhage from stomach and bowels.  
Death.*

UNDER DR. SPENCER.

Isaac I., æt. 40. 1 day.

Patient, who was a Chinaman, and could not speak English, was brought to the Infirmary, suffering from pain in bowels and sickness. No history could be obtained. The pains appear to have come on the night before admission. He had vomited altered blood. When seen he seemed to be in great pain, rolling about and pressing on his abdomen; skin bile-stained; very little liver dulness; slight ascites. He moaned a great deal. Shortly after admission he vomited up some altered blood, and also passed blood, less altered, per anum. The pain continued very severe, the hæmatemesis recurred several times, and he got gradually weaker, and died about six hours after admission.

*P. M. E.*—Typical “hob-nailed” liver, very small. some slight serum in peritoneal cavity. Slightly altered blood in stomach and intestines. The ruptured vessel could not be found. Cicatrix of old gastric ulcer, but this did not seem to have anything to do with the hæmorrhage. Kidneys firm in consistence. Lungs emphysematous.

## CASE XXVI.

*Chorea. Ascarides in intestines. Bronchitis. Death.*

UNDER DR. SHAW.

Mary Ann I., æt. 16. 30 days.

Patient had had a previous attack of chorea about

five years before, which she thought was induced by a fright. Had never had rheumatism. Had been rather unsteady for nearly two months. She was a well-nourished and well-developed girl. Jerking affected all her limbs to such an extent that she could not walk, nor could she feed herself or help herself in any way. Her head twitched very much. Facial muscles constantly working. Speech jerking and indistinct. Quiet during sleep. No cardiac bruit was heard on admission, but a systolic blow heard at the base two days after. She had some difficulty in swallowing. She was treated first with chloral and bromide of potassium, but without any good effect. Ice-bag to the spine, hypodermic injections of curara, and afterwards of morphia were tried, but without any better result. The morphia caused arrest of the respiration, with alarming symptoms. She got rapidly worse. The jerking became much more violent, and she got very little sleep. A bed-sore formed over the sacrum, and extended deeply. The cardiac bruit varied in character and intensity from time to time, but could generally be heard. The pulse became weak and compressible. She lost control over her sphincters. Nutrition became greatly impaired. Swallowing became so difficult that she had to be fed by enemata. She became quite unable to articulate, and could not protrude her tongue when asked to do so. Cough came on about a fortnight after admission, and she had great difficulty in expectorating. Stimulants were given freely, as ammon. carb., sumbul, and brandy. As sedative drugs had scarcely any effect on her, the administration of chloroform was begun, and continued for three days, keeping her lightly under its influence for four or six hours at a time. This kept her quiet, and the chorea gradually diminished, until the jerking left her

almost entirely. Unfortunately, however, her bronchitis increased, and her temperature rose considerably. On the day of her death she vomited up two ascarides, and passed another from the bowel. A few hours before her death her temperature rose nearly to 106° F. She was then put into a bath at about 90° for a few minutes. This lowered the temperature, but no permanent improvement took place. She gradually sank, and died exhausted.

*P. M. E.*—Brain was hyperæmic, but no other pathological appearances were found. There was a circle of beady vegetations on the mitral valve. Lungs congested, œdematous, and bronchi plugged with mucus. Three or four ascarides in intestines. Recent intussusception of bowel. No peritonitis.

#### CASES XXVII. AND XXVIII.

##### *Two cases of Guinea-worm.*

##### UNDER DR. SHINGLETON SMITH.

F. H., æt. 37, seaman.

Cruising off Gold Coast for twenty-two months; never went on shore; drinking-water brought to ship by natives, and no other water drank. Three months after return from cruise admitted to Infirmary. Ten days before admission noticed a hard and somewhat painful swelling in right buttock; in a week it discharged "moisture," and a worm protruded; during efforts to extract, the worm broke; swelling, pain, and discharge increased; marked constitutional symptoms. Three days before admission a similar swelling appeared in the left upper arm, with similar phenomena, but no attempt made to extract the worm. On admission both swellings discharging a milky



fluid, and a worm protruded from the one in the arm; a good deal of this worm was withdrawn; under microscope the milky fluid was like pus with numerous, motionless young filariæ. Subsequently several swellings appeared on trunk and limbs; no worms found on incision; they pursued same course as the first. The swellings were all treated by poultices and incision; the worms, when they could be got at, were wound out gently round a piece of wood, and in one case the entire worm was recovered. General treatment by assafætida, quassia, and cascarilla. The wounds healed slowly.

A shipmate of the above also came under observation. In this case the symptoms and course were much the same; severe constitutional and local symptoms followed breakage of the worm during an attempt to withdraw it.

The natives of the Gold Coast are strongly of opinion that the worm is introduced into the body through drinking-water.

#### CASE XXIX.

*Varicose condition of cutaneous abdominal veins from obstruction of vena cava. Improvement.*

UNDER DR. SHINGLETON SMITH.

John D., æt. 39. 27 days.

About seven years ago patient received a blow on the back from a heavy tub of coals while at work. He lay down for about two hours, and was then able to resume his work. A few hours afterwards he passed a considerable quantity of blood from the bowel. He did not feel any other ill-effects from the blow, and continued at his work as usual for the next three months. He then had an illness which compelled him to give up work and go

into Hospital. He says he had shiverings and some pain in back and legs. He was in Hospital for seven weeks, and while there he noticed that the veins in the left side of his body were becoming enlarged. Shortly after leaving Hospital the left leg became swollen and very painful. This continued for some time, and then it went down again, but has always been liable to swell a little after exertion. Right leg became swollen and painful about a year after the left. Has been in Infirmary once before and an out-patient several times. Has suffered from cold feet usually and occasional attacks of severe cramp in abdomen. Ten weeks before admission he had pain in loins, which extended down right thigh and leg and that limb became swollen, red and tense. On examination, the superficial veins of left leg and left side of abdomen and chest were found very much dilated, tortuous and hard. Less marked on right side. Right leg and thigh swollen, tense and painful. No cardiac disease. He was kept at rest in bed, sedative lotions applied to the right leg, and iodide of potassium given internally. The swelling of leg went down very much, and he was able to return to work again.

## CASE XXX.

*Syphilitic laryngitis. Extreme dyspnœa. Tracheotomy. Temporary relief. Death from intercurrent pneumonia.*

## UNDER DR. SPENCER.

Daniel O'L., æt. 28. 34 days.

Patient had had syphilis about two years before he came under observation. He had suffered from cough, dyspnœa, and huskiness of voice for about three months, gradually getting worse, when he came into the Infirmary

for the first time. He was treated with potass. iod., blisters, &c., and went out greatly relieved. He was again admitted, about ten days after, with extreme dyspnoea, his face being very much cyanosed. Tracheotomy was recommended, but he refused to submit to it. He was kept in bed and treated with potass. iod., &c., again, and for some time he improved considerably. About three weeks after admission he again became very bad, but still declined operation. The dyspnoea became so bad, however, that he became quite asphyxiated, and the operation was then performed. He was almost quite unconscious when it was done. The relief was immediate, and in a short time he was comparatively comfortable. He went on very well for a week, his temperature remaining below normal. He then had a severe rigor, the temperature ran up to 103° F., and the physical signs of a severe attack of right pleuro-pneumonia became developed. He grew rapidly worse, and died on the fifth day. No *P. M.* allowed.

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## Obituary.

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CROSBY LEONARD.

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WITHIN the last few months we have lost an old and dear friend and colleague, and it is but right that the first number of our Reports, in which he felt much interest, and to which, had he lived, he would no doubt have contributed, should take notice of the event; and this we propose to do in a brief account of his life in the order of time and as concerns us chiefly from a professional point of view; and we hope to be excused if, in performing this labour of love, we become discursive, and yield now and then to an inevitable tendency to slide backwards into the past whilst writing of our Alma Mater.

And how is it that the members of our profession have disappeared so rapidly from the scene of late, in Bristol and Clifton? The place, healthy beyond most places for others, seems fatal for us. The House Surgeon of ten years ago is Senior Surgeon to-day. Do we neglect the laws of life we lay down for others, being, as

has been said before now, like the sign-post, pointing out the road to the temple of Hygeia, but never travelling along it ourselves, or is it the incessant labour of a trying and anxious profession, in which work is very great and rest is very little, and in which we bear the burden of other persons' anxieties in fighting for them the battle between life and death, and in which we also have to suffer, to an extent incredible to the inexperienced, the unreasonable worries and caprices of some of our patients ?

The causes, whatever they may be, seem to be local, and we trust only temporary, so that a little later on there will be seen again in our consultations and at our meetings grey heads, with active brains and healthy bodies, giving, as heretofore, their experience to appreciative juniors.

CROSBY LEONARD, an only son, was born on the 16th of May, 1828, at the corner of Brunswick-square, Bristol, where his father, Mr. Isaac Leonard, carried on a very considerable general practice for many years. His mother's maiden name was Crosby, and the family belonged to the Baptist denomination, which had their head-quarters at the Academy in Stoke's Croft and in the old chapel in Broadmead, to which about that time numbers were attracted by the eloquence of Robert Hall. His early education was at a school for little boys kept by the Misses Overbury, in Ashley Place, which he left for the Bristol College, entering first into the "Junior Department" at a very early age in the year 1838. The College, although young in years, was even then beginning to decline; and the head-master, the Rev. J. E. Bromby, left it shortly afterwards and established a school at Mortimer House, Clifton, but Crosby Leonard

remained and completed his school studies at the Bristol College.\*

Of his school days there is but little to be said. One of the masters of the Junior Department, Mr. John Exley, remembers him as a good and industrious little boy, and as he began so he seems to have gone on. To judge from some extant "Certificates of Honour," which were awarded to boys who just failed to carry away the prize in their class, he got on fairly with most subjects, and as one of these certificates was for "Proficiency in Natural History," with the date of December, 1838, he must have had a peculiarly early taste for studying the subjects cognate with his future profession. He does not appear to have been much addicted to athletic games, which, indeed, were not encouraged as they are now, nor were the opportunities so many or so great, although the love and enjoyment of them were the same.

His preliminary education was not pushed very far, and in June, 1844, at the early age of 16, he made the formal entry into the outer circle of the profession by being apprenticed to his father for five years, according to the excellent custom in vogue at that time.

After spending rather more than a year in this intro-

\* The old Bristol College was established in the year 1830, in a high house at the bottom of Park Row, where the Jewish Synagogue has since been built. It had a very prosperous career of little more than twelve years, and seemed exactly to satisfy the want felt in Bristol at that time, sending many boys to Oxford, who obtained Scholarships and afterwards Fellowships at Trinity, Balliol, Oriel, Corpus and Magdalen, and to Cambridge, where they turned out Scholars, and Fellows, and Wranglers, and First Class Classical Tripos Men; it had among the boys a Senior Wrangler, Professor Stokes, of the Royal Society; it had the late President of Trinity College, Oxford, one, if not two, of the Indian Lawrences, Walter Bagehot, Mr. Justice Fry, and many others who have risen to the top of their several professions in after-life; and the influx of a number of first-class University men, who were masters of the College, gave a very definite stimulus to the literary society of the city at that time.



ductory work, which consisted in learning the art of dispensing and the uses of drugs, and the pharmacopœia, with the small amount of chemistry requisite for that purpose, in acquiring also the art of bleeding and cupping adroitly, with the usual preliminary trials at tooth drawing, and in becoming somewhat familiar with anatomical and medical terms, he entered the surgical practice of the Bristol Infirmary (for it had not then its royal cognomen) on the 1st of September, 1845, as a pupil of Mr. Lowe, the Senior Surgeon, for three years, the first of which was spent as a “walker;” and in October of the same year he began his attendance in the first year’s lectures at the Bristol Medical School, entering as general perpetual pupil.

Those were the days of long and elaborate courses of lectures: every pupil had to attend three winter courses of Descriptive and Surgical Anatomy and of Anatomy and Physiology, and in all the three years when Mr. Leonard attended at the school one lecturer gave the whole of the three courses of Descriptive and Surgical Anatomy, each course consisting of one hundred and forty lectures of one hour each, and the lecturers on Medicine and Chemistry and Materia Medica had each to give one hundred lectures in the winter session.\* It is not too much to say that Mr. Leonard was one of the most regular and punctual and attentive pupils that ever worked at the Bristol Medical School; as far as the lectures on Descriptive and Surgical Anatomy are concerned an extant record shews that he attended nearly every lecture, and that he also worked well and closely in the dissecting room.

\* In this winter—1845-46—the lectures on the Theory and Practice of Medicine were given by Dr. Symonds, Dr. Bernard, and Dr. Budd; being the last time that the two former lectured, and the first appearance as teacher of William Budd.

At the end of his first year's surgical practice as walking pupil he began his duties as dresser, going "into the house" as surgical pupil of the week for the first time at the end of September, 1846.

The working of the Infirmary and the special status and work of the dresser of the week were different from what they are now. Of clinical instruction there was practically none; the older class of surgeons, of whom Mr. Lowe was the last typical specimen, seemed never to entertain the idea of teaching; at that time few words of information or explanation were heard in the wards, and no remarks were made after consultations and operations; and of the surgeons in office at the time we write about, viz., Mr. Lowe, Mr. Harrison, Mr. Morgan, Mr. Hy. Clark and Mr. Green, our present Senior Consulting Surgeon was the only one who broke through the routine of silence, and gave occasional clinical lectures, until the College of Surgeons made it imperative some years afterwards.\* And the dressers, a fairly numerous body, were expected to attend all operations, and generally got a word of reprimand if they did not come down in good time to operations at night and in the evenings, and they had to arrange with the boys to call them when they took out the notes to summon the surgeons. And when the

\* A very few years earlier than the time we write about, when there was but one qualified resident officer, the House Surgeon, a great deal of the teaching in the medical wards devolved upon him, and many of the pupils were accustomed to go round with him in the mornings when he made his notes of the cases; and on this account one of the physicians, who resigned very shortly before Mr. Leonard's time, for many years received none of his pupils fees, and he always had a fair share of pupils, but handed them over to the House Surgeon. The Infirmary held the anatomy license, and unclaimed bodies were kept and dissected in the summer months, as many as three being dissected sometimes at once: this was not very long after the passing of the Anatomy Act and the instinct of having to provide your own subjects for dissection had hardly died out.



physicians had seen their out-patients in the mornings, the dresser of the week had to “do the bleedings” and cuppings which had been prescribed, the number varying from one or two to upwards of twenty, so that the old bleeding room had at times a very unpleasant aspect, and later in the day he had to bleed and cup in the wards and make setons or issues if any had been prescribed. Until the appointment of a second legally qualified man in 1843 as the House Apothecary (called Assistant House Surgeon some years later), the dresser of the week was the *locum tenens* of the House Surgeon when he went out, the theory being that the House Surgeon always left word where he was to be found. The house pupils claimed and exercised the right to make all the medical *post mortem* examinations, and the facilities for getting specimens of healthy or diseased organs for study or for making preparations were great, and the students freely availed themselves of them.

Having finished his three years as surgical pupil, he entered in October, 1848, to the medical practice under Dr. Lyon, the Senior Physician of the Infirmary, for eighteen months, the time then required by the Apothecaries Company; and at the same time he began an attendance at Mr. Estlin’s Dispensary for the cure of Eye Complaints, which, with the small exceptions of absence from Bristol or from illness, he continued regularly as long as he was able to do any work at all.

He obtained the Supple Medical Scholarship and gold medal in this the first year in which it was awarded, having already carried off the second and third years’ prizes at the Medical School, ending thus honourably his career as student in Bristol. Mr. Greig, who was House Surgeon at that time, writes, that “Leonard was one of



our best pupils. \* \* Conspicuous for punctuality and also for kind consideration for the poor patients, many of whom were warmly attached to him."

About this time, as a senior student and as one of the honorary secretaries, he took an active part in bringing about a "Bristol Infirmary Dinner," the only one within the memory of man, when the medical and surgical staff and the present and old pupils dined together at the Montague Tavern, and Mr. Leonard's methodical and business-like habits did good service in carrying out the arrangements. Mr. Lowe, the Senior Surgeon, occupied the chair, and there was a large company; but the proceedings were the reverse of lively. The stewards were Alfred Bleeck, John Bishop Estlin, William J. Goodeve, Frederic Granger, John Harrison, Isaac Leonard, Gilbert Lyon and Edward Waldo, of whom two survive, namely, Mr. Harrison and Mr. Granger.

In December, 1849, he went up to London, and within a day or two passed the examination for the membership of the Royal College of Surgeons, at that time a single examination on one evening in anatomy, physiology and surgery, and returned to finish his medical practice, which he did, and "passed the Hall" in June, 1850.

At the end of the year he went to Paris, where he seems to have been busily employed for some time, attending the medical and surgical practice and the courses of operative surgery on the dead subject at Clamart and the Ecole de Médecine, which could not be done in England at that time, and he also attended the private cliniques for minor surgery and eye diseases.

He joined, as did almost all the English students at Paris, the Parisian Medical Society, where he read a paper on a case of trephining for epilepsy, which had

occurred in the practice of Mr. Henry Clark at the Bristol Infirmary.

Early in 1851 he returned to Bristol and started in practice with his father.

From one of Mr. Leonard's note books it appears that he made short notes in a tabular form of all the operations he saw from a very early period, and we see classified under different heads a vast series of cases, and it is certainly a sign of great industry as well as of considerable power of arrangement that he began thus early on his first entering a surgical practice, and kept to the same system until he had himself for some years been Surgeon to the Infirmary.\*

Upon one or two occasions he resided at the Infirmary in the place of the House Surgeon and Assistant House Surgeon when they were away upon their holiday.

Mr. Leonard soon began to take a part in public medical matters, and an enumeration of the many and various appointments he held in Bristol will be almost his professional history during the rest of his life.

At the time we are writing about, viz., 1851, the Bristol Royal Infirmary Medical Society was in existence, one of several similar bodies which had flourished there for a time as long as some unusually active secretary managed its affairs, and then died of inherent lack of nutrition. Mr. Leonard attended the meetings and read some papers there, one on a case of Cesarean Section he had seen in Paris under Dubois, and another upon the symptom "Insensibility," and he became Demonstrator of Anatomy,

\* Among the operators named are Goldwyer, Estlin and Nat. Smith, of Bristol; Stanley, Lloyd and Sir W. Lawrence, of St. Bartholomew's; and Partridge, Bowman and Sir W. Ferguson, Nelaton, Civiale, Jobert, Roux, Velpeau, Guersant, Sichel, Maissonneuve, Vidal, Cullerrier and Paul Dubois.

his first connection as teacher with his old school, in 1853. In this year also he was elected the Bristol Secretary of the Bath and Bristol Branch of the Provincial Medical Association, by which means he was brought into contact with most of the profession in Bristol and the neighbourhood, and his pleasant manners and address and habits of business made him a very popular secretary, under whose guidance the Society flourished.

Mr. Leonard obtained at this time two other public appointments, which had a definite influence on his private practice. He was made Honorary Surgeon Accoucheur of the Bristol Dispensary, a post which he held for nine years; and some months afterwards he was elected to a similar position in the Bristol Lying-in Institution, which he held for rather a longer period. These appointments meant that he was to be called by the attendants to all unnatural and specially difficult cases, and thus he had, and availed himself of, a vast field of observation and experience in this particular branch; so much so that his manual dexterity and good judgment and practical skill were not surpassed, if they were equalled, by those of any practitioner in the neighbourhood, for, in addition to these qualifications, he possessed coolness and patience and firmness, which inspired great confidence in the patient and her friends, and it is not to be wondered at that as his general practice increased the obstetrical part of it increased yet more rapidly.

In 1854 Mr. Prichard resigned his share in the lectures on Descriptive and Surgical Anatomy, and Mr. Leonard was appointed as his successor, dividing the course with Dr. Brittan, and he held this Lectureship for ten years, having Mr. Coe as his colleague for the greater part of the time. He was a steady, systematic and



popular lecturer, and a pleasant colleague to work with, and the Bristol students going up to London for their examinations maintained the character they had long held of being "well up" in their anatomy.

When the Surgeoncy to the Bridewell became vacant in March, 1854, Mr. Leonard was the successful candidate for the post, which he held for nearly twenty-four years. This was a paid appointment, involving a daily visit to the prison and the charge of a most uninteresting set of patients, but connected with it was the responsible duty of examining and certifying for the lunatics before the magistrates; and when his health failed and he gave up this work they accorded him a vote of thanks and a small annual pension. These offices necessarily brought him into frequent intercourse with the City authorities, and he thus had a large share of inquest cases requiring *post-mortem* examinations and opinions to give upon medico-legal matters. A year or two afterwards he became Assistant Surgeon, and then Surgeon to the Bristol Eye Dispensary, performing his duties there with great attention and his usual regularity. He was an expert and successful operator for cataract and other cases of operations in the eyes requiring care and nicety.

The next public appointment which Mr. Leonard obtained was in 1859, when he was commissioned as Assistant Surgeon to the 1st Gloucestershire Rifle Volunteer Corps. The Volunteer movement was young, and we were not so accustomed as we are now to see our civilian friends in the garb of soldiers; so it was a surprise to his colleagues, and no doubt a gratification to the patients, when, after his election to the Infirmary, he first appeared in the wards on Saturdays, in uniform ready for the march out. In this, as in all other duties,

he was regular, punctual and attentive; but promotion was not very rapid, for it was not until 1871, upon Mr. Hore's death, that he got the step onward and was gazetted full Surgeon.

Mr. Isaac Leonard died in 1859, after an illness of considerable length, in which he felt great comfort in the efficient care of a skilful and unwearied and affectionate nurse; and his death, besides putting all the practice into his son's hands, where indeed it had been for some time, left a vacancy in the Medical Reading Society, which he had joined in the early part of the century, and to this Crosby Leonard was elected as soon as the usual formalities of the society had been carried out. He continued his membership for twenty years, until within a very short time of his death.\*

In the early part of the next year, 1860, Mr. Harrison resigned the Surgeoncy of the Bristol Royal Infirmary, having been elected in 1836, at a time when there had been no vacancy for twenty years, Mr. Nathaniel Smith having been Junior Surgeon all that time.†

\* It would be out of place and of very little general interest to say much about a private reading society, yet, because of its antiquity and because at one period it was almost exclusively an Infirmary Society, consisting only of members of the staff and old Infirmary pupils, and to remember some of the old names of those with whom Crosby Leonard had some acquaintance, we may be permitted to allude to it. It was established about 1807, and among the early members were Mr. Estlin, Dr. Stock and Dr. J. C. Prichard, Mr. Hetling and Mr. William Swayne (house-surgeon), and Mr. J. C. Swayne and Mr. Leonard, some of whom were members for more than forty years; it always followed as nearly as possible in its original track, being limited to twelve members, for the usual purpose of circulating medical books. They meet once a month and dine together once a year in the January meeting, and usually spend a pleasant evening, ending with a sale of books. At the dinner in January, 1859, some of the members remarked that the presence of an invited stranger brought the number up to the fatal thirteen, and the ideas of the superstitious were thus confirmed.

† The rule restricting the term of service to twenty years was made in 1843, and was altered subsequently, so that the only members of the faculty who came

The hope of obtaining this, the highest surgical position in Bristol, as usual, brought several competitors into the field, with more or less idea of success, for it seems to be allowed that these elections are legitimate opportunities in which young men may, without offence against professional usage, advertise their acquirements in the public papers. Our legal friends certainly have the advantage of publicity over us: all their graver operations are performed in open court; the instruments are named, opened out and explained, and the whole case, with the names of the operators and their assistants and the consultants, together with the result of treatment, is laid before the public the next day.

In 1857, when Mr. Hore was elected almost by acclamation, for he had been a very popular House Surgeon for many years, Mr. Leonard, Mr. Metford, Mr. T. E. Clark, Mr. James Prowse and Mr. Eubulus Williams put in an appearance, and in 1860 similar steps were taken by Mr. Ormerod, Mr. Metford, Mr. Mortimer Granville and Mr. T. E. Clark, all old pupils and eligible, but Mr. Leonard distanced them all, and by the present system of proxy votes the matter was virtually settled before the day appointed, and soon the new Surgeon to the Infirmary made his appearance in the operation-room as a skilful operator.

under its influence, and were therefore obliged to resign, were Dr. Fairbrother and Dr. Fox on the medical and Mr. Green and Mr. Prichard on the surgical side. In 1850, upon the death of Mr. Lowe, who was elected in 1807, Mr. Harrison became Senior Surgeon, to the great comfort and advantage of the staff under him. He was a first-rate surgeon, almost the first advocate in this country of a tonic and strengthening plan of treatment after operation, in which he was particularly adroit and successful; but besides his professional talents, and others which do not concern us here, what most struck the writer of this notice was his great discretion and sound conciliatory advice in his capacity as our chairman whenever any untoward event, or some specially delicate matter occurred between the surgeons and the pupils, or the Committee or the public.



The only noteworthy event that concerns our story in the next two or three years was his removal, in 1863, from Brunswick Square to Rockleigh House, Clifton, where he spent the rest of his life. The neighbourhood of the large squares in the eastern part of Bristol had changed very much for the worse since Mr. Isaac Leonard first lived there: the merchants and professional men and others of the chief citizens who inhabited those mansions had obeyed the usual law in large towns and had journeyed westward, and Mr. Leonard was constrained to follow the fashion. And many changes had occurred among the staff of the Medical School, but Mr. Coe and Mr. Leonard were still at this period the lecturers on Descriptive and Surgical Anatomy until 1864, when Mr. Prichard resigned the Chair of Surgery which he had held since 1849, at first with Mr. Henry Clark and as sole lecturer for three years, and Mr. Coe and Mr. Leonard were appointed in his stead, while their anatomical course was undertaken by Mr. T. E. Clark and Mr. Lansdown. Mr. Leonard resigned his share of the surgical course in 1871, when the late Mr. Tibbits became Mr. Coe's colleague.

It will be imagined from what has been written that Mr. Leonard had by this time his hands pretty full of business, and few, if any, of us have ever held so many and such varied professional offices at the same time, for besides his private practice, which had greatly extended since his removal to Clifton, and all these appointments which we have already enumerated, he was Surgeon to the *Formidable* training ship, to the Baptist College, and to various schools and factories, which brought him in more or less continuous work. He seemed, however, equal to the occasion, and with the exception of frequent

and severe headaches, which did not appear due to overwork, he generally appeared able to get through his day's duties comfortably.

In the year in which Mr. Leonard gave up his connection with the Bristol Medical School, 1871, more honours dropped upon him.

As has already been said, he was at this time gazetted full surgeon to the Rifle Corps, and in the course of the summer the sudden death of Mr. Ralph M. Bernard\* gave him his promotion by making him Senior Surgeon to the Bristol Royal Infirmary, a responsible position any surgeon might be proud of holding, for besides taking the chair at all consultations, upon him depend in great measure the order and punctuality of work on the surgical side, and Mr. Leonard's business habits and firm and kindly disposition were exactly the qualifications required.

In the same year, too, he was elected President of the Bath and Bristol Branch of the Medical Association, the highest honour we can give to any member of the profession here; he succeeded the late Charles Bleek, of Warminster, a popular, liberal and hospitable president. Mr. Leonard's presidential address was, as might have been expected, a sound and sensible one, alluding to the members who had died in the past year, among whom was Dr. Symonds; and speaking of the advantages of the Society and other usual topics of this kind, and afterwards more especially treating of Bristol sanitary matters, of public medicine, of the registration of dis-

\* Mr. Bernard, who was elected in 1857, in the vacancy caused by the retirement of Mr. Morgan, was a surgeon of the old school, rather after the type of Mr. Lowe, whose pupil he had been. He was an extremely useful member of the surgical staff, being regular and punctual and a strict disciplinarian, so that not only the pupils but also the resident officers felt his influence. His life came to a sad and abrupt end, for he fell over a cliff in South Wales, in the presence of his wife and children, and was killed instantly.

ease, and the spread of contagion ; and his year of office passed peaceably and pleasantly away with the usual hospitalities, which, beginning in a casual way as extraneous adjuncts of the young society, had at last grown and become part of the actual organism itself.

In 1873 he became Fellow of the Royal College of Surgeons of Edinburgh, and he now seemed to have attained in the professional way all that he desired, in fact all that he could possibly get in honours or preferments.

Yet he worked on, hard and industriously, with real love of his professional duties, for some few more years, until increasing weakness of his muscular system, which left his mental powers intact, made him gradually give up one occupation after another. He had increasing difficulty in walking, and, although in the greater part of his practice he walked about the first half of the day, using his carriage only in the second half, and thus got through on foot some miles a day, he for many years appeared to be laboured in his gait, and to find the exercise which he thought necessary to his health irksome and trying to him.

In the end of the summer of 1875 he fell down in his own garden and broke his leg, and was never so well after this time. His case did well and he afterwards got about fairly, but having been somewhat stout and heavy before his accident, he became more so during the period of his confinement to bed, and this rendered the latter part of the treatment of his fracture more tedious and difficult.

His hand and leg became after a time heavy and numbed, and being reduced first to his carriage, which he mounted latterly with excessive difficulty, and then to his house and afterwards to rooms on the same floor upstairs,



it appeared evident that the disease in his spinal cord, whatever it was, was making progress; but the account of his illness is written elsewhere in this book by abler hands. In 1878 he sent in his resignation to the Infirmary Committee, and received a vote of thanks for his careful and assiduous work, and was made Consulting Surgeon. Mr. Arthur W. Prichard was elected Surgeon in his place, and Mr. Board became Senior Surgeon.

Whilst he was confined to his two rooms upstairs he still saw his old friends, and seemed much to enjoy a chat about medical matters, or any medical gossip that was afloat, or changes in the medical world, or about any remarkable case in the neighbourhood, and at such times and we believe generally, he was cheerful and always took his full share in the conversation, and now and then he would see one of his old patients who much wanted his opinion, and to the last he was, mentally, as capable of advising as ever he was. He was, however, continually becoming more disabled and feeble, and having been spared any confinement to his bed, which would have been most irksome and painful to him, he died quietly and peacefully and without pain, but somewhat suddenly, on the evening of Monday, the 13th of October, 1879, at the comparatively early age of 51.

Such was Crosby Leonard's professional life, and we think it was a happy one, in which incessant work was rewarded by continuous success. He died a very wealthy man, for at the age when many medical men first begin to feel less need of the perpetual strain and struggle of their earlier years, he had already amassed a fortune, which for a surgeon in practice has, in these parts, scarcely ever been equalled or approached.

Mr. Leonard was never married, but lived at home

quietly with his sisters, all of whom survive him. He was fond of society, and was always able and willing to take his part in conversation upon any subjects, and, with his pleasant manners and good temper, was a general favourite.

He took a holiday occasionally, and being an excellent sailor, always preferring a stiff breeze and a rough sea to one more suited to the majority of landsmen, was accustomed to take short trips across to Ireland, or to Liverpool and Belfast, or up the Irish and Scotch coasts, of which he saw a good deal; and with the comfortable proviso of an entire absence of sea sickness, a medical man, wearied with his work or worried by bad cases, and possibly with some little malarious taint just beginning to poison him, would regain more health in a short time by this kind of change than any other. A very few summers ago he went the trial trip in the *Polly*, the tender brig to the *Formidable*, in which, as the captain told the writer, he was one of the very few, if not the only one, who stood the lively and stormy night off Ilfracombe without alarm or illness.

In politics he belonged to the liberal party, but was not an ardent nor violent politician.

In his work he was a careful observer, very attentive and considerate in the examination of his patient and accurate in diagnosis, and in consultation always ready with his reasons in support of his opinion. He was not anxious for operations if they could be avoided by other or safer treatments, but when an operation was decided on he was always prompt and active, and while he was sufficiently expeditious, he was watchful and equal to any of the emergencies that so often turn up in the course of severe or dangerous operations; in fact, he felt the confi-

dence in himself which sound anatomical knowledge and a good strong nerve imply. He was ready to try new remedies when they came with the sanction of names on which he could rely, and new instruments and appliances whenever they shewed reasonable hope of being improvements on the old ones; but he did not himself originate any changes of importance, nor was his name often seen as a contributor of cases or articles in the Medical Journals.

In his general practice he was held in much esteem and affection by his patients, as those of us know who have attended any of them since his death, and as was testified on the day of his funeral, when not only his own family and private and professional friends and the medical students, but a large number of his former patients met at Arno's Vale to take the last opportunity of shewing their respect and love for him.

His professional intercourse with his colleagues was conducted with the strictest integrity. He was a gentleman by birth and education and instincts, and we never heard the slightest hint of any act of his, professional or otherwise, unworthy of the name, for to act according to the acknowledged laws of medical etiquette is, in other words, to act as an honest man and a gentleman.

He was particularly deficient in the art of quarrelling. He had, no doubt, his share of troubles and anxieties; he had his exacting and dissatisfied patients and his strongly opposed opinions to those of others; he had his impracticable and restless colleagues; his unreasonable and impenetrable subordinates, and many of the other worries of professional life, as we all have in varying degrees, but with him it never led to anything approaching a rupture or quarrel with his opponent, so that in these respects



the tenor of his way flowed more evenly with him than with some of us. He was, in fact, to a rare degree kind, and good, and true, and placid; and, without attempting to gauge the loss which his family has met with, we must admit that his death has made a peculiar gap in the ranks of our profession which it will take a very long time to fill up.

A. P.

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## WILLIAM BUDD, M.D., F.R.S.

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THE words written in the beginning of the account of Mr. Leonard's life have been speedily confirmed by the death of Dr. William Budd, Senior Consulting Physician to the Infirmary. Although he had attained a good age in comparison with many who had gone before him, his work had been for some years finished; and few as were these latter and painful years of his life, the rapid change and promotion before referred to sufficed to introduce a medical and surgical staff into the Infirmary, who, with one or two exceptions, had no personal acquaintance with our distinguished colleague. The principal medical events of his life are given in the following short narrative, for the greater part of which we are indebted to the kindness of his brother, F. N. Budd, Esq.

He was born in September, 1811, at North Tawton, in Devonshire, where his father carried on for many years a large general practice. He was the fifth of nine sons of this talented family, of whom seven became physicians, and five graduates of Cambridge, being all of them Wranglers, and four of them Fellows of their Colleges; four also graduated at Edinburgh, and two were Fellows of the Royal Society, namely, the subject of this notice

and his brother, Dr. George Budd, late of King's College Hospital, who, with five others, still survive.

William Budd never went to school, but was educated at home until he was sixteen years old, when he went to Paris and completed the routine of his general education by attending the lectures at the College Royal de France.

His medical work at Paris was under Louis, Andral, Cruveilhier and Broussais, with some of whom he was well acquainted; and whilst studying there he was taken with typhoid fever, and treated so actively by leeching and starving, according to Broussais' system, that, although he ultimately recovered, he was obliged to give up his reading and other work for several years. After an attendance at the Middlesex Hospital as a pupil of Dr. Watson, he entered the University of Edinburgh, where he graduated in 1838, carrying off the gold medal of his year for his inaugural thesis on rheumatic fever.

In London he became intimate with Watson, Buck and Paget, and in 1840 he succeeded his brother, Dr. George Budd, as physician to the Dreadnought hospital ship, where, curiously enough for one whose fame rested to a considerable degree upon his researches into this particular disease, for the second time he contracted typhoid fever, which again nearly proved fatal to him.

In 1841 he came to Bristol and settled in lodgings in Park-street, in the house in which Dr. W. B. Carpenter, of physiological renown, had previously established himself, where he first started in practice with the orthodox brass plate and "Surgeon" upon it.

To come to Bristol and to begin practice as a physician was an up-hill fight at first, especially for one who



had no special introductions in the place, and it was many years before Dr. Budd had established himself securely and satisfactorily in general practice; but he was not idle, for he joined and took an active part in the meetings of the Provincial Medical Association and in the various scientific societies, having been one of the founders of the Bristol Microscopical Society and the Pathological Society, which latter has recently come to life again under the name of the Bristol Medico-chirurgical.

He soon made himself known as an active member and clear debater at these meetings, and on the advent of the cholera in 1849 he was appointed one of the members of the various committees organised at that time by the Pathological Society to investigate the disease in all its aspects, and on that occasion, it will be in the memory of some of our older members, we believed that we found peculiar bodies, or “cholera germs,” in the ejecta from the cholera patients, as well as in the air contaminated by their presence; and the discussions on this matter, in which Dr. Budd had an active share, were carried on with much interest and a good deal of *animus* in the medical journals and also in the public papers of the day.\*

During all this time Dr. Budd kept steadily in view the great object of his ambition—the appointment as Physician to the Infirmary. He saw at first large numbers of poor patients gratuitously at his own house; and in the year 1847, only six years after coming here, he was elected Physician to the Infirmary, without much opposition, in the place vacated by the late Dr. Riley. In his

\* The *Lancet* and *Medical Times* of 1849 contain some of these articles, with wood-cuts of the microscopical appearances, and the *Morning Chronicle* and other London papers had leading articles and long communications upon the subject.

hospital work he was zealous and enthusiastic, on one occasion describing to the writer of these lines how much happier his life was now that his occupation and powers of usefulness were so much increased, and saying that, as he walked down, for he had not then attained the dignity of a carriage, as soon as he got within sight of the Infirmary, like a boy within view of his bathing-place or cricket-field, he could hardly restrain himself from setting off to run, in his anxiety to see how his cases were getting on.

Dr. Budd had published before this time his paper on the "Symmetry of Disease," and many others in the *Lancet* and other medical journals, and was the President of the Bath and Bristol Branch of the Association in the year 1855-56, when he took for the subject of his presidential address "Longevity and the normal duration of life in man;" and being appointed to read the Address in Medicine at the Annual Meeting of the British Medical Association in Bristol in 1863, under the presidency of the late Dr. Symonds, he chose as his theme "Variola Ovina and the laws of Contagious Epidemics, illustrated by an experimental type."

He was elected Lecturer on Medicine at the Bristol Medical School in 1845 as colleague of Dr. Symonds and Dr. James Bernard, and resigned in 1854, during which time he had as colleagues Dr. G. D. Fripp and Dr. Stanton, in one session giving the entire course himself.

In 1862 Dr. Budd resigned his post as Physician to the Infirmary, and was made Consulting Physician; his practice having largely increased during the latter part of the period when he held office. He went frequent distant journeys, and saw daily at his house many patients from

all parts of the neighbouring country, besides his own ordinary and consulting practice in Clifton and Bristol, working at the same time in every spare half hour at the book on typhoid fever, which he contemplated bringing out and actually finished, although at the time of its publication his health had finally broken down; and with this strain upon his mental powers he was so overdone that he was obliged to take occasional periods of complete rest, in Switzerland or elsewhere, in order to recruit his health, and he would return to his duties apparently well for a time, and then would break down again, until between six and seven years ago his powers failed him, and he lingered on a complete wreck of body and mind, both of which had a few short years before shewn the greatest vitality and energy, and he died near Clevedon early in January of this year.

Dr. Budd left a widow, and a family of two sons and several daughters.

When in the prime of health he was a most enthusiastic lover of his profession, the principal object of his regard; and in his practice, whether they were Infirmary or private patients, he took the most anxious care of all in his charge; and whatever else he took up he undertook with all the energy of a sanguine temperament, whether it was some scientific investigation or sketching or painting or taking photographs, in all of which pursuits he shewed considerable skill, and which he amused himself with until the increase of his practice absorbed too much of his time and thoughts. He was subject to occasional intense headaches, possibly the result of his fever of former days, and without doubt the precursors of the attack which ultimately proved fatal.

Dr. Budd got through a great deal of hard work of a



very good kind, some of which will prove a valuable and permanent addition to our ever increasing stores; and although this is not the place for a critique or special report of his writings, the following imperfect list, with which we close this notice, will bear evidence to his industry and the variety of subjects he wrote about, and in their choice we notice throughout an evident bias towards questions of contagious diseases, their natural history and modes of propagation and prevention.

To the Medico-Chirurgical transactions he contributed articles on the Pathology of the Spinal Cord and Gout in 1839, besides the paper on Symmetrical Diseases before referred to, and in the weekly medical periodicals the following long and varied series of papers communicated occasionally up to the year 1869:—The Frog as a detector of Tetanic Poison—Mode of Propagation of Cholera—Fever at the Clergy Orphan Asylum—Intestinal Fever and its propagation—Intestinal Fever essentially Contagious—Intestinal Fever, its propagation and relation to Sewerage (1859)—Disinfection—The Prevention of Infectious Diseases—Kousso—Distended Gall Bladder—American quack advertisements—Cholera, its cause and prevention—Bronzed Skin—Propagation of Cholera—Dr. Snow and the Board of Health—Cholera at West Ham—Diphtheria—Pythogenic theory of Typhoid—Scarlet Fever and its prevention—Contagion of Yellow Fever—Typhoid and the Board of Health—The Propagation of Malignant Pustule—Typhoid of Cattle—Propagation of Phthisis—Bacteria and Malignant Pustule—Variola ovina—The Investigation of Epidemics—The Cattle Plague—Typhoid and its prevention—Hydatid Cyst of Liver—Sugar and Diabetes—Calcareous Salts in Rickets—The Pathology and causes of Cancer—Purpura

and the styptic properties of turpentine—Cholera in Bristol—Croup—Cholera—Scarlet Fever—besides the address on Medicine in 1863 and his book upon Typhoid Fever published just at the beginning of his last illness in 1873.

A. P.

## HOSPITAL STAFF.

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### *Consulting Physicians.*

Dr. BRITTAN, Dr. FAIRBROTHER, Dr. E. LONG FOX.

### *Consulting Surgeons.*

Mr. HARRISON, Mr. AUGUSTIN PRICHARD.

### *Physicians.*

Dr. SPENCER, Dr. SHINGLETON SMITH, Dr. WALDO,  
Dr. SHAW.

### *Surgeons.*

Mr. BOARD, Mr. DOWSON, Mr. ARTHUR PRICHARD,  
Mr. F. R. CROSS, Mr. GREIG SMITH.

### *Assistant Surgeon.*

Mr. W. H. HARSANT.

### *Medical Superintendent.*

Mr. J. H. L. MACINTIRE.

### *House Surgeon.*

Dr. LENDON.

### *House Physician.*

Dr. WATSON.



## INFIRMARY PRIZEMEN, 1878-79.

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1878.

*Medical Supple Prize and Medal.*

CECIL HENDERSON } equal.  
ARTHUR H. BOISSIER }

*Surgical Supple Prize and Medal.*

GEORGE MUNRO SMITH.

*Clark Prizeman.*

GEORGE MUNRO SMITH.

1879.

*Medical Supple Prize and Medal.*

GEORGE MUNRO SMITH.

*Surgical Supple Prize and Medal.*

J. PAUL BUSH.

*Clark Prizeman.*

FRANK S. PECK.

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*The "Tibbits" and "Leonard" Scholarships will be open  
for competition next year.*

PATIENTS ADMITTED AND DISCHARGED DURING 1878.

IN-PATIENTS.

Remaining on the Books on December 31st, 1878, to be carried to the Statement for 1879	...	...	...	207
Remaining in Wards on December 31st, 1877, and since discharged	...	...	...	206
Fresh Cases admitted from December 31st, 1877, to December 31st, 1878	...	...	...	2124
Total Cases treated during 1878	...	...	...	2,330
Average daily number of Patients	...	...	...	218.28
Average number of days in the House	...	...	...	33.72

GENERAL RESULTS.		MEDICAL.	SURGICAL.	TOTAL.
Cured	...	228	289	517
Made Out-Patients, Convalescing	...	270	442	712
“ Relieved	...	169	97	266
Relieved	...	78	55	133
Unrelieved	...	16	10	26
Went out for various reasons	...	56	42	98
Died	...	107	58	165
Remaining in Wards on December 31st, 1877, and since discharged	...	79	127	206
Remaining in Wards December 31st, 1878	...	85	122	207
Total Results—In-Patients		1088	1242	2330





PATIENTS ADMITTED AND DISCHARGED DURING 1879.

IN-PATIENTS.			
Remaining on the Books December 31st, 1879, to be carried to the Statement for 1880	...	...	244
Remaining in the Wards on December 31st, 1878, and since discharged	...	...	207
Fresh cases admitted from December 31st, 1878, to December 31st, 1879	...	...	2561
Total number of Cases treated during 1879	...	...	2,768
Average daily number of Patients	...	226.10	
Average number of days in the House	...	32.56	

GENERAL RESULTS.		MEDICAL.	SURGICAL.	TOTAL.
Cured	...	178	497	675
Made Out-Patients, Convalescing	...	345	532	877
“ Relieved	...	218	128	346
Relieved	...	81	60	141
Unrelieved	...	12	19	31
Went out for various reasons	...	39	41	80
Died	...	102	65	167
Remaining in Wards on December 31st, 1878, and since discharged	...	78	113	191
Remaining in Wards on December 31st, 1878, and since died	...	7	9	16
Remaining in Wards on December 31st, 1879	...	100	144	244
Total Results—In-Patients	...	1160	1608	2768















